THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. VII. JANUARY, 1927. No. 27.

Original Papers.

OBSERVATIONS ON EPILEPSY.*

By WILLIAM ALDREN TURNER, London.

Epilepsy is a very old disease. During many years there has accumulated around it an enormous literature and it is quite impossible in a short paper to do more than touch upon one or two of its aspects.

It is well to remember that recurring convulsions stand in close relation to many internal medical diseases, to disease and injury of the nervous system and to mental disease. Epilepsy also has its psychological side and there is reason to believe that there may be some relation between it and endocrine disorder or deficiency. Moreover it presents a social problem of some magnitude on account of the large number of cases requiring permanent care in special institutions.

While recent work has thrown new light upon it I do not think it can be claimed that the secret of epilepsy is yet revealed.

The essential feature of epilepsy is a disturbance of consciousness, occasional, sudden, swift and brief. This may or may not be accompanied by spasm or convulsion. Hughlings Jackson[1] long ago associated with this disturbance certain psychical phenomena—the so-called 'intellectual aura'—such as dreamy states, a feeling of reminiscence, double consciousness, feelings of being somewhere else, or as of something impending, visual hallucinations and so on; all such sensations being transient, in some cases preceding the fit, and at other times actually forming the attack itself.

"There are all degrees," he says, "of severity of epileptic paroxysms, from giddiness attended by trivial confusion of thought to a full violent seizure with universal convulsion and deep coma." To this we might also add, as evidence of epilepsy, certain twitchings of the muscles and myoclonic symptoms localised to a limb or segment of the body, precedent to some fits and not necessarily accompanied by loss of consciousness.

*Presidential address delivered before the Section of Psychiatry, Royal Society of Medicine, December, 1926.

There has been a tendency during recent years to broaden the basis of epilepsy mainly from the psychological side and to regard as epileptic states of disturbed consciousness of longer duration and occurring occasionally, such as some fugues, somnambulisms, and stupor states, temporary phases of mental confusion and sudden maniacal outbursts. Rows[2] in particular has laid stress on this broadening of the conception of epilepsy and has stated that epilepsy may now be regarded as the prototype of every functional mental illness. Whether this idea leads to a greater clearness regarding the nature of classical epilepsy of which a fit is the outstanding and spectacular feature, may be the subject of difference of opinion, but at all events it brings into line certain phases of disturbed consciousness attributable to emotional causes and reveals the difficulty which exists clinically in distinguishing certain so-called 'hysterical' reactions on the one hand, and some periodic mental or psychical states on the other.

It would be desirable to restrict the term 'epilepsy' to those occasional, sudden, short and swift losses or disturbances of consciousness, with which this term has been associated for so long a time, and to regard as 'equivalents' those longer phases of disturbed consciousness which have been brought within the ambit of this disease. It is one of the features of this disorder that the characteristic periodic and transient disturbances of consciousness may be observed in association with different fundamental causes. Hence epilepsy is 'symbolic' and occurs under circumstances in which there may be evidence of organic cerebral disease or injury, in some toxic or infective conditions, and as a sequel of emotional distress.

It is now generally accepted that there is no single clinical entity to which the name of epilepsy may be applied. There are, on the other hand, many epilepsies, and there would appear to be accumulating evidence that the epileptic type of fit reaction may result from mental as well as from physical causes.

From a consideration of the main facts of epilepsy, it would seem as if three special groups may be distinguished; they are the organic, the toxi-infective or metabolic, and the psychogenic, while the great mass of residual epilepsy may in the meanwhile be designated as 'epilepsy of unknown origin.'

Epilepsy of Organic Origin.

It is generally accepted that any cerebral organic lesion may cause, as one of its symptoms, seizures having the features of an epileptic fit. It is not necessary to refer in detail to these organic conditions, but they include all forms of cerebral vascular lesion, not excepting the arteriosclerotic degeneration of late life with its senile epilepsy, taboparesis and paralytic dementia usually in the earlier stages, cerebral tumour and destructive lesions of the brain following penetrating wounds of the skull.

It has been recognised for a long time that a cerebral new growth may give rise, as one of its symptoms, to seizures differing in no respect from those occurring in epilepsy of unknown origin. When epileptic attacks are present in association with the other general symptoms of intracranial tumour, no difficulty is experienced in forming an opinion of their symptomatic character; but the type of case to which I now refer is that in which generalised epileptic seizures occur over long periods of time—sometimes for several years before symptoms indicative of the new growth make their appearance. It is difficult to ascertain just what is the frequency of this group of epilepsies. If it were possible to follow through for long periods more of our epileptic patients, it might be conceded that cerebral tumour was possibly a more common cause of recurring fits than is generally accepted, more especially of that variety which begins in adult life. These cases are not infrequently diagnosed and treated as epilepsy on account of the periodic recurrence of both the major and minor type of fit, the absence of any localising signs, and the often definite arresting influence on the fits of the bromide salts.

Such cases are more likely to occur in late adolescence or early adult life. In my series the average duration of the epilepsy before the obtrusion of general symptoms was about four years, although in one case the fits were those of an ordinary recurring epilepsy lasting for sixteen years, and cases have been recorded for longer periods. The frontal and temporosphenoidal lobes are the two regions of the brain affected by tumour which are most prone to show evidence of generalised epileptic fits. Certain distinguishing features may occasionally be detected in the type of seizure, by which the site of the lesion may be diagnosed; but in the by no means frequent absence of such special evidence, signs which would indicate an organic epilepsy of this nature are a well defined local aura, the presence of some degree of post-convulsive weakness tending to become more persistent, a permanent bilateral inequality of the deep reflexes, and an extensor plantar response during the interparoxysmal interval.

'Traumatic Epilepsy.'

By traumatic epilepsy is meant a disability characterised by seizures having the features of ordinary generalised epilepsy, occurring as a late phenomenon in consequence of a wound or injury of the skull or brain. The outstanding feature of this condition is the development of the disease after a latent period of several months sometimes, up to two or three years, following the trauma, and the absence of any definite or constant relation between the injured region of the brain and the method of onset of the major fit. The fits resemble those of ordinary epilepsy and all varieties may be observed from a momentary 'absence,' lapse or vertigo, to the fully developed major seizure. The disability runs a chronic course and in the majority of cases some degree of mental enfeeblement tends to supervene.

This disability is entirely distinct from focal or Jacksonian epilepsy following head trauma. The latter condition is not epilepsy in the proper acceptation of the term; rather it is a temporary state of localised cortical irritation. It is observed within a short time of the infliction of the trauma; it is more amenable to surgical interference; it shows a tendency often towards spontaneous cure, and once the attacks have ceased, there does not seem to be any special tendency to relapse, or to the development of traumatic epilepsy[3].

The generally unsatisfactory outlook in traumatic epilepsy is due probably to the fact that the seizures are not attributable solely to the local effects of scarring or cicatricial adhesions about the cortex and subcortical tissues, but are evidence of an epilepsy in which the local destructive lesions play only a part once the epilepsy has been established. Moreover, only a relatively small percentage of cases (about 5 per cent.) of penetrating wound of the head develop epilepsy, an important observation which would lead to the conclusion that something other than local tissue changes at the seat of injury is requisite for the production of a traumatic epilepsy.

In these two forms of epilepsy, as indeed in all forms of gross cerebral lesion with seizures of an epileptic kind, there would seem to be a common factor which would explain their occurrence. What this factor may be is just one of those problems which underlie all epilepsy. One would expect this common factor to be of vascular nature and to be dependent on some periodic temporary and local disturbance of the cerebral circulation (A. E. Russell[4]). McRobert and Feinier[5] have attempted to explain the frequent recurrence of generalised fits in tumours of the temporosphenoidal lobe by local pressure of the tumour upon the sylvian artery and the effect of this pressure upon the arterial supply and stability of the cortex so affected. But this can be only one example of the gross circulatory disturbance within the cranium, which must accompany all cerebral tumours. Sargent[6] has described the extensive cerebral lesions found in gunshot wounds of the brain—contusions, inflammatory softenings and cicatrices binding the scalp to the damaged brain, and has noted also the striking rapidity with which visible circulatory changes may occur in the cortical circulation in response to various disturbing causes.

It is impossible to believe that the serious and extensive tissue-changes associated with tumours and traumatic lesions are unattended by gross local alterations in the cerebral vascular supply, and it is easy to assume that local circulatory disturbances may have a potent effect in the causation of the seizures in all forms of organic epilepsy.

But, as already pointed out, this factor alone will not explain all the facts of symptomatic epilepsy. It has been stated by Myerson[7], whose work on inheritance will be referred to later, that it is unnecessary to invoke a predisposition in epilepsy when a properly placed lesion of the brain can by itself

produce the disease. The facts of traumatic epilepsy seem to refute this attitude, as I can confirm what has been pointed out by others, that only a small percentage of cases of penetrating head wounds or of cerebral tumour develop epilepsy.

Just what are the circumstances attending the initiation of the major epileptic fit, whatever the fundamental cause may be, is one of those problems on which there has been much speculation. There are, however, some direct observations of the cortex during an epileptic fit. Foster Kennedy[8] described how he observed the brain of a patient during a general fit through exposure of the parietal cortex in the performance of an operation. "The initial sign," he says, "was a sudden whitening of the cortex, which was no sooner remarked on than it was replaced by venous engorgement with protrusion of the brain beyond the level of the bone defect. This was coincident with the tonic stage of the attack and the period of general clonic convulsion."

But the question naturally arises, (1) what is the cause of the vascular spasm? And, (2), are the vascular changes and the cortical anæmia produced thereby the cause or the consequence of the epileptic movement?

If the vascular spasm and the cortical anæmia are the initiators of the fit, what factor exists in certain persons to give rise to periodic, sudden and transitory attacks of this kind often persisting throughout a lifetime? If these vascular phenomena can be established as the prime movers in the fit, is it not more likely, as suggested by some writers, that the inherent instability in epilepsy may be found in the sympathetic or vegetative nervous system, rather than in neural instability?

Fraser[9] and others have attempted to show that in consequence of a local cerebral lesion (which is visualized also in idiopathic epilepsy) a toxin is evolved, having the features of the histamine group of poisons, and acts as an irritant upon the cerebral blood vessels and leads to sudden, local and spreading anæmia of the cerebral cortex. But, it may be asked again, what is the evidence of such a condition? Or what would explain a periodic, sudden and temporary toxæmia arising unexpectedly in what is often seemingly good health?

And in this connection a further problem arises. Assuming a vascular mechanism in the initiation of the grand mal, does a similar condition occur in petit mal—those momentary blanks, 'vertiges' or 'absences' which are so characteristic of some epilepsies? Moreover, is this vascular instability also the cause of the longer states of disturbed consciousness described as 'epileptic equivalents'?

Epilepsy of Metabolic Origin.

A proposition has been put forward that all the phenomena of epilepsy may be explained by the presence of toxic agents circulating in the blood,

acting upon the musculature of the cerebral blood vessels, and leading to vaso-constriction and convulsions. These toxic substances, it is alleged, may be derived from various sources, such as defective carbohydrate metabolism in the small intestine (Cuneo), from overaction of the adrenals in states of emotional distress, or from the breaking down of nervous tissue locally in cases of cerebral tumour, cerebral trauma and other organic disorders of the brain.

J. S. Collier[10], an advocate of the toxic metabolic theory of this disease, gives the following reasons in favour of such a basis rather than any condition of cerebral instability or functional derangement. The remarkable periodicity of the seizures; their association with certain anaphylactic causes such as pregnancy and the puerperium; the incidence of the status epilepticus and its association with fatty degeneration of the myocardium, and the immunity of the habitual epileptic from many of the common diseases. He is of opinion also that the bodily and mental make-up common to so many confirmed epileptics, the mental deterioration and the terminal dementia, are evidences of a metabolic error, either by way of deprivation or of long continued poisoning.

There can be little doubt that some cases of epilepsy at the commencement of the illness, and some complications or developments during the disease, are attributable to toxi-infective or metabolic agencies. Of this character may be enumerated outbursts of serial epilepsy and of the status epilepticus. These are states of acute or subacute epilepsy ushered in by well-marked premonitory signs, characterised by fits recurring with great frequency and severity over a limited period of time, and separated by more or less prolonged intervals of freedom; often associated with an accidental circumstance during the course of the disease, such as a sudden stoppage of bromide medication, the puerperium, or an acute inflammatory disorder. Other types probably falling under the same grouping are those which ensue during or within a brief interval of the exanthemata and other common infective disorders. In eliciting the histories of patients with recent epilepsy I have been impressed by the frequency of otitis media and mastoid disease shortly antecedent to the first seizure. Operations for appendicitis also figure as an occasional determining cause of the first fit. Of possibly like nature are the epilepsies arising *de novo* in or aggravated by pregnancy and lactation, as also those developing out of a puerperal eclampsia.

It has been suggested that in that variety of the disease known as the 'menstrual type,' a chemical influence is at work through the agency of ovarian hormones. It has been shown also that a form of epilepsy may be associated with intestinal stasis and the toxæmia resulting therefrom.

The relation between epilepsy and disturbance of endocrine glandular function is too uncertain to admit of discussion here. A quite unproven degree of importance has been attached to it by some writers, although there is evidence of a relation between pituitary tumours and epilepsy, and cases have been seen in which from other evidence, it is probable that the normal endocrine

balance was impaired. It may, however, be stated that therapeutic measures based upon endocrine deficiency have, in my experience, been attended by entirely negative results. There would appear also to be a small and uncommon group of epileptic reactions of an anaphylactic type, depending upon a hypersensitivity of some persons to certain food poisons, or who show a susceptibility to certain forms of diet.

Such are some of the clinical facts bearing upon a toxi-infective or metabolic group of epilepsies. But if asked whether there is biochemical support of this proposition, it has to be admitted that such evidence is scarce, and when it exists, is not convincing.

The biochemistry of epilepsy has been the subject of much investigation, but of the methods of study it has not been fruitful of definite results. Certain fairly constant findings have been noted by some observers, but in the bulk of the cases the changes in the blood would seem to be the direct effect mainly of the nervous and muscular disturbances consequent upon the fit. The cerebrospinal fluid would theoretically offer the best field for biochemical changes bearing upon this disease, but in practice its analysis has given almost consistently negative results.

The position, therefore, as regards a metabolic group of epileptics is that considerable clinical evidence exists in its favour, but that so far convincing biochemical evidence is lacking.

Is there an Epilepsy of Psychogenic Origin ?

Most treatises on epilepsy refer to the determining influence of excitement, fright, shock and anxiety in the causation of fits. Emotional causes have come to be regarded as of great importance in psychogenesis, but it is only within recent years that mental exploration has revealed how emotion acts in the causation of mental illness, and the clinico-psychological study of epilepsy has revealed a group of epilepsies, or at least of epileptic reactions, which would appear to be attributable to emotional influences.

Most of us have seen cases in which a shock or fright has given rise to seizures of an epileptic type, while in other cases it has been difficult to say whether the attack was epileptic or hysterical.

In this connection we are at once confronted with the difficulty of distinguishing the hysterical from the epileptic types of fit, and every practitioner knows how difficult this is especially when the attack is associated with an emotional or psychical cause.

It is known also how often the hysterical type of reaction may merge into one having epileptic features ; and the conjunction of the hysterical and epileptic type of reaction may be observed at different times in the same person. Alan McDougall[11] states that fits occur that are epileptic in appearance, but hysterical in origin, and that there seem also to be fits that are hybrids of epilepsy and hysteria.

Although major hysterical attacks are, as a rule, fairly distinct in character from major epileptic seizures, yet the latter not uncommonly may show a 'hysterical' colouring. The minor attacks of both disorders are often not easy to differentiate. Not infrequently minor attacks, often regarded as functional, hysterical or of little consequence may recur for years before a major epileptic fit ensues and gives the clue to their real nature. In cases of doubtful nature Yealland[1,2] states that the simple method of reproducing the fit by asking the patient to raise his legs against resistance will confirm the diagnosis of hysteria.

There is no doubt also that some persons are liable to attacks which cannot be classified either under hysteria or epilepsy. These attacks are in the main purely subjective, but in some cases, objective signs of a circulatory and vasomotor character are observed. On the subjective psychical side they may be characterised by a sense of fear, dread or apprehension, and although consciousness is not abolished, the mental state may assume a dreamy attitude associated with a feeling of unreality. Such attacks have been placed upon the borderline of epilepsy and in the majority of cases are associated with emotional causes.

Similarly on the psychiatric side it is practically impossible to distinguish, in certain stupors, fugues and somnambulisms, the hysterical from the epileptic, and it is equally difficult to diagnose a sudden acute attack of mental confusion with violence—a so-called 'equivalent'—from a temporary post-convulsive confusional outburst.

Now the factor common to all these conditions is some disturbance or loss of consciousness and in the view of Rows and Bond[2] the study of the various types of epileptic attack has demonstrated that some emotional state, involving a disturbance of consciousness, and some reaction to express the emotion have been found in every instance. In their view also the similarity between the disturbances of consciousness in epilepsy and other functional nervous attacks has not been sufficiently recognised. To quote their words: "The revival of a memory depends on the activity of similar mechanisms whether it gives rise to the aura which precedes the epileptic seizure, or whether it occurs as what is termed a hallucination. A wild outbreak, whether it is seen in an epileptic, a maniac, or a hysteric, results from the activity of similar mechanisms in each case. Fugues and dreamy states, whatever be the form of mental illness of which they form a part, have a similar origin. The epileptic is separated from the others only by the occurrence of the seizure, which although the most dramatic, is by no means the most important part of the disturbance of consciousness."

In the opinion of these writers the evidence is so conclusive that they hold that even major epilepsy is mainly the result of a disturbance of consciousness produced through memories and the emotions associated with them.

It is generally accepted that the sympathetic autonomic system is disturbed by emotional stimulation and activity. Such disturbance, it is assumed, may have indirectly an action upon the peripheral bloodvessels; and if the view already referred to that vasoconstriction is a factor in the initiation of the epileptic attack is accepted, there is here a possible explanation of the emotional or psychogenic type of epileptic reaction. But here again we are confronted by one of the big unsolved problems of all epilepsy, and in this case the question is whether even an intensified emotion, without any other associated factor, is sufficient to give rise to that dramatic crisis—the major epileptic fit, with its lightning-like onset, its profound loss of consciousness, its tonic-clonic convulsion, and its grave post-convulsive phase in which even life itself sometimes appears to be threatened.

The whole subject of the epileptic fit being the expression of an emotional state involving a disturbance of consciousness requires fuller examination. The patients who were studied by Rows and Bond were war cases, and we know from other evidence that the war neuroses stood on a somewhat different plane to those commonly met with in civil life. Some evidence which these writers bring forward in support of their view is based on the benefit which their patients derived from psychotherapy; but there is reason to believe that the psychoanalytic method of treatment of epilepsy has not had that beneficent effect which its advocates had anticipated.

It seems to me that in this, as in all other forms of epilepsy, an additional factor has to be looked for and will most probably be found in a constitutional defect obtained through inheritance or acquired later on.

My own experience in this matter is in favour of the view that psychical or emotional causes may give rise to seizures differing in no respect from those of a generalised epilepsy. These may be the determining agency not only of the first fit, but are frequently the active cause of individual seizures in the course of the confirmed disease. There is another aspect of this subject, for we know by experience of epilepsy that the prevention or postponement of a fit is not always desirable. In some instances the fit is so definitely a reaction to a difficult situation, which the patient is unable to meet normally, that its occurrence clears the air and a feeling of relief is the outcome. The view that there is a psychogenic variety of epilepsy may not be generally accepted, but there is accumulating evidence to this conclusion. The relation between emotional distress, hysteria and epilepsy has been summarized tersely by an anonymous writer in the *Lancet*, who says[13]: "Attacks of idiopathic epilepsy following on emotional strain and stress have been observed. The student is compelled to recognise that certain patients exhibit seizures clinically indistinguishable from ordinary epilepsy as reactions to situations of a purely psychical nature. In these patients, dream states, outbursts of temper, excitements and even maniacal attacks recur periodically on the heels of emotional experience. The emotional nature of the original experience is no

criterion of hysteria. It will be seen how the horizon of epilepsy widens and what justification there is for the contention that a psychogenic basis for the epileptic fit should be sought out, or at least should not be ignored, because the patient bites his tongue or is incontinent."

The Epileptic Mentality.

The epileptic mentality has been described by many writers on this disease. It is rare indeed to find habitual epileptics who do not present some degree of mental obliquity. They are usually self-opiniated and egotistical; moody, with periods of laziness and lethargy, alternating with outbursts of hastiness and pugnacity. They are difficult to live with and are often useless as workers. The majority are unable to adapt themselves to social conditions and as a result they become self-centred, morbid and asocial.

Pierce Clark[14] defines the epileptic constitution as characterised by a "personality defect of egotism, morbid sensitiveness, hypochondriasis and poverty of ideas." In consequence, he states, the epileptic is incapable of social adaptation and is rendered inadequate to lead a normal adult life. This is present from earliest childhood and may be studied in a detailed psychological investigation of the life-history antecedent to the onset of the fits. When present in an outstanding degree, its possessor may develop into an established epileptic.

The existence of this mental 'make-up' in the potential epileptic has been criticised. In the first place such a mental endowment, when found, is not necessarily associated with epilepsy; and, secondly, is it a correct description of the mentality as observed before the onset of epileptic seizures?

I have investigated the temperamental qualities of a large number of young epileptics seen in private practice, and the results do not confirm what has been described as the 'epileptic personality.' The absent-minded, the indifferent and the day-dreamer formed only a small number of the cases. Those who were described as passionate and difficult to manage were about equal in number to those whose mentality was bright, intelligent and often precocious. The self-satisfied, selfish and self-opiniated formed quite a small group. On the other hand the outstanding feature was that which may be described as nervous, anxious-minded and easily worried. These young persons were often highly emotional and were described by relatives as "neurotic" and sometimes hysterical. A small number were found to be reticent, studious and reserved. Such types indeed, might be found amongst a similar number of young persons in any school.

Rows[2], also, in his recent work on epilepsy, denies the existence of a mental 'make-up,' such as has been described as characteristic of the potential epileptic. He states that the varieties of disposition amongst them are as great as amongst other people; they may be gay or sad, irritable or submissive, self-assertive or

lacking in self-confidence. If there is any constant feature it is probably one of anxiety, and the repetition of the emotional states associated with the seizuresleads to a habit of mind and body, which is termed the epileptic mental 'make-up.'

An increasing knowledge of the psychology of epileptics convinces me of the great part played by anxiety, apprehension and fear in the development of their mental outlook. The uncertainty as to when or where a fit may come on breeds anxiety and apprehension. Some seizures, more especially of the petit mal variety, are accompanied by an intense sense of fear. Long ago Hughlings Jackson described fear as a symptom associated with an epigastric warning, and most writers on epilepsy have called attention to this striking phenomenon, which may even actuate the patient's conduct after an attack.

There are so many features of the disease of an alarming kind, that, as it progresses, an increasing degree of anxiety fills the mind of the patient and determines his behaviour.

How often has one observed young people who, prior to the development of their epilepsy, have been regarded as normal, but who, owing to the occasional and uncertain occurrence of a fit and probably also from the restrictions placed upon their occupations, pleasures or recreations have developed the belief that they are abnormal, have become sensitive to criticism and as time went on, have tended towards asocial habits.

All writers on epilepsy are agreed as to the mental enfeeblement, sometimes amounting to dementia, which may develop in the chronic epileptic and there is a general consensus of opinion that this mental crippling results from the repetition of seizures over long periods of time.

Further, I would submit that the acquired nature of the epileptic mentality in many cases, receives confirmation, if or when an opportunity is offered of observing an arrest of seizures even after the disease has lasted for many years. In a few striking cases of arrest of fits by aid of luminal therapy, after years of illness, I was able to observe the passing away of the anxieties and difficulties of the epileptics' existence, the re-establishment of self-confidence and a return to a more normal social life.

Epilepsy of Unknown Origin.

After cases have been referred to one or other of the groups already mentioned, and this obviously can be done in many instances only by the lapse of time, or after prolonged individual study, a great mass of undefined epilepsy still remains. To this mass the term 'epilepsy of unknown origin' may tentatively be applied.

Although there is no age period during which epilepsy may not occur, those groups just described are rare before adult life. The most common epoch for the onset of the so-called idiopathic disease is between the ages of

ten and twenty. It would, therefore, seem as if the common epilepsy of puberty and early adolescence was associated with the development and growth of the nervous system and the coming to maturity of the organs of reproduction, and the instincts arising therefrom. It is during this epoch that quite insignificant, or apparently insufficient causes such as infections, fatigue, overwork and minor degrees of trauma, may light up the epileptic tendency in some persons: while in others no determining cause can be detected even after careful investigation.

In many young persons the onset of the first fit is so often, as it were, 'a bolt from the blue,' that astonishment is expressed at its occurrence in conditions of good physical health. According to the psychoanalytic school this happening may be explained on the view that the fit is the expression of some unconscious need, craving or conflict. Although it is conceivable that circumstances of this character may precipitate an epileptic reaction in certain peculiarly constituted individuals—those possessing what has been described as the 'epileptic personality'—it is impossible in the present state of our knowledge to accept such a view as applicable to more than a limited group, what may be described as a 'temperamental group' of epileptics.

Amongst many features of interest in the clinical study of epilepsy a few only may be mentioned. I have been impressed by the not uncommon onset of the disease in normal young people during a period of rapid physical growth at about 16 or 18 years of age. This observation would suggest that a too rapid growth of the nervous tissues concurrently with the ordinary process of physical development may be a determining factor in certain cases.

Another observation of interest is the relatively frequent occurrence of epilepsy in only children. I do not know whether there is any causal connection in this association, but it is interesting to recall the statement of St. Luke, when describing the miracle of the cure of epilepsy, that the sufferer on that occasion was an only child. Whatever its bearings may be the observation has long tradition behind it.

There is a type of epilepsy characterised by the occurrence of a few fits temporarily at some period of life, or at such wide intervals as to make it hazardous to connect them. The question arises here whether such cases as these should be regarded as epilepsy, or merely as reactions of an epileptic kind consequent upon some temporary and non-recurring cause. Several examples of this type have been observed in adults and it is just possible that with present knowledge, they might be included within the psychogenic group, or may have been explainable by a temporary metabolic upset.

Another group of much interest is that in which a few 'faints' having the features of le petit mal may occur during puberty or early adolescence, and no recurrence takes place until the advent of arteriosclerosis in late life determines the onset of the major type of fit.

In striking contradistinction to these varieties of epilepsy, is that group in which the disease, arising in later childhood or at puberty, develops rapidly; the fits increase in frequency and severity, mental enfeeblement ensues and in a few years the patient's condition is reduced to that of an epileptic dement. The explanation of this type of case is probably to be found in structural changes in both the nervous and neuroglial elements of the brain, secondary to the frequent recurrences of severe major fits. These structural changes may be found post mortem in all diseases in which convulsive seizures have been a feature during life.

Inheritance.

A few words may be said about inheritance in epilepsy, as there has been a tendency during recent years to comment adversely upon and to underestimate the importance of the hereditary element in this group of illness. The predisposing influence of heredity has been the subject of enquiry by all writers on epilepsy and statistical tables of an elaborate, but not always satisfying kind, figure in the old treatises. It may be true that in these tables sufficient distinction has not always been made between the several groups of epilepsy, as now revealed, and a too wide range of so-called hereditary, but sometimes irrelevant factors, has been included within the ambit of statistical enquiry. It may well be that the old methods of investigation are untrustworthy and crude in the light of modern knowledge and that evidence on this matter should be sought for along biometrical lines. But it is impossible to survey the family history of any large number of epileptics without obtaining direct evidence of 'epilepsy in the family' in quite a substantial percentage of the cases.

Recent figures of my own from 250 cases seen in private practice revealed a percentage of 32 per cent. with a family inheritance of epilepsy. In a previous series[15] of 890 epileptics, 37 per cent. gave a family predisposition to this disease. Russell Brain[16] has recorded his observations and enquiries into this subject in 200 hospital cases and found that 28 per cent. had a family history of epilepsy. Of 80 patients with a family history of epilepsy in my recent series, 33 per cent. came of an epileptic parent and a further 9 per cent. had an epileptic grandparent: epilepsy in parents or grandparents therefore being noted in nearly half the cases.

From the above facts it is difficult to dispute the existence of a family predisposition to epilepsy in a certain number of epileptics, approximately about 33 per cent. of all cases. Indeed, I would go further and admit what may be entitled a 'familial group' of epilepsy—a group in which the disease may be traced through several generations. How else can one explain such examples as the following: epilepsy in a brother and sister, their mother and a maternal uncle: epilepsy in two brothers, whose grandfather was

epileptic ; an epileptic patient whose aunt, uncle and cousin also suffered from epilepsy ; a patient whose father, and paternal grandfather were epileptics ; and another whose father, grand-aunt and grand-uncle were epileptics ?

The alleged infrequent occurrence of epilepsy in brothers and sisters has been urged by Myerson[7] as evidence against a hereditary influence, but this occurrence is not so rare as might be supposed. Russell Brain amongst his 200 patients found 24 families with two or more affected siblings and in my series of 250 cases, 20 families had two or more siblings affected with epilepsy.

Although we accept the existence of a hereditary predisposition in some 33 per cent. of all cases of epilepsy the cause of this disability has still to be ascertained in the remaining 67 per cent. This large group, in which no traceable heredity to epilepsy is found, forms indeed the common group encountered in ordinary practice. Myerson has contended also that epilepsy is an affair of the individual and not of the stock and that it represents injury, perhaps to a germ-plasm, whether that injury be pre- or post-natal. His contention is that there is a constitutional factor in these cases, which may arise *de novo* from intrauterine life onwards. In this group sometimes the most severe and intractable cases of epilepsy are found. Parental alcoholism, causing injury to the germ-plasm, may account for a small number ; in my cases only six per cent. being accounted for in this way. Certain continental authorities, however, give a much larger percentage due to this cause.

In other instances evidence may possibly be available, although difficult to obtain, of the existence of a dyscrasia, an infective disorder, an intoxication or even local disease of the germ-forming organs, or in the mother during the period of intrauterine life of the child. But these possible causes are mainly in the realm of conjecture.

It is just here that again we are brought face to face with another of those at present unsolved problems of epilepsy.

What is the cause of epilepsy in so large a percentage of cases without traceable heredity and without evidence of organic, metabolic or psychogenic foundation ? What is the evidence, if any, of injury to the germ-plasm ? Developmental anomalies of structure are found in the brains of epileptics, but similar changes may be found with other diseases and even in the brains of normal persons. Are there any means by which neural or vasomotor deficiency or instability may be detected, except by inference, after epilepsy has declared itself ?

These are questions to which it is difficult to supply an answer. It is, however, sufficiently apparent that a large number of human beings have some defect, obtained either through heredity or acquired during or after intrauterine life, whereby the organism reacts harmfully to certain endogenous conditions, to cerebral trauma, to stress or strain, to infections and to emotional causes.

BIBLIOGRAPHY.

[1] JACKSON, HUGHLINGS, *Brain*, 1881–1898.
[2] ROWS and BOND, *Epilepsy*, London, 1926.
[3] TURNER, ALDREN, *Jour. Neur. and Psychopathol.*, 1923, iii, 309.
[4] RUSSELL, A. E., *Lancet*, April, 1909.
[5] MCROBERT and FEINIER, *Jour. Amer. Med. Assoc.*, 1921, lxxvi, 500.
[6] SARGENT, P., *Brain*, 1922, xlv.
[7] MYERSON, *Inheritance of Mental Disease*, Baltimore, 1925.
[8] KENNEDY, FOSTER, *Arch. Neurol. and Psychiat.*, 1923, ix, 567.
[9] FRASER, D., *Clinical Studies in Epilepsy*, Edinburgh, 1924.
[10] COLLIER, J. S., *Brit. Med. Jour.*, 1924, ii, 1045.
[11] MCDOUGALL, ALAN, *Lancet*, 1920, ii, 764.
[12] YEALLAND, *Lancet*, 1923, ii, 651.
[13] *Lancet*, 1919, ii, 791.
[14] CLARK, PIERCE, *Clinical Studies in Epilepsy*, Utica, 1917.
[15] TURNER, ALDREN, *Brit. Med. Jour.*, 1910, March and April.
[16] BRAIN, RUSSELL, *Quart. Jour. of Med.*, 1926, 299.

SOME RESULTS OF A SECOND INDUCTION OF MALARIA IN GENERAL PARALYSIS OF THE INSANE.

By J. ERNEST NICOLE, and
JOHN P. STEEL, Warrington.*

INTRODUCTION.

Malarial therapy has been used in this hospital on all suitable cases of general paralysis of the insane for the past three years. During the last eighteen months it has been thought justifiable to induce pyrexia for the second time in those cases where insufficient improvement was evidenced after the first attack, and where the physical condition was satisfactory.

In so far as experience has shown us the slowness with which mental improvement may take place after the first attack of malaria, a renewal of the treatment has not been considered advisable until some considerable time has elapsed after the termination of the first attack, and an accurate assessment of the mental condition has been possible. In fact, the average length of time between the first malaria and any subsequent inoculation has been fourteen and a half months: owing to lack of success in our early endeavours malaria only actually occurred on an average seventeen months after the primary attack.

In all, twenty-five cases have been deemed suitable for further malarial treatment, and on these fifty-two inoculations were performed (see Table). On the first twenty cases our own well-established strain of infected blood was used, but it was effective in only five instances; the remaining fifteen being subjected to inoculation on several occasions without success.[1]

Three of these 'resistant' cases were then exposed to mosquito infection; one patient showed a few parasites in the peripheral blood but developed no pyrexia. The other two had true malaria, and one of these acted as donor to other 'resistant' patients.[2]

Following the introduction of this mosquito strain eleven of these previously 'resistant' patients developed satisfactory rigors. Five others were then re-inoculated for the first time and in all except one the procedure proved successful.

It will be seen, therefore, that twenty patients have had induced malaria for the second time.

---

*From the County Mental Hospital, Winwick, Warrington.

Case No.	Health.	Habits.	Inoculated.	Improvement.		Habits.	Reinoculation.	Improvement.	
				Mental.	Physical.			Mental.	Physical.
3	Fair	Good	10 Jan., 1924	Nil	Nil	Good	29 Apl., 1925	—	—
8	Poor	Faulty	11 Apl., 1924	Slight	Marked	Good	27 July, 1925	—	—
							17 Dec., 1925	Marked	—
16	Poor	Faulty	27 Feb., 1924	Slight	Marked	Good	4 Mar., 1926	Nil	Nil
17	Poor	Faulty	11 Feb., 1924	Nil	Slight	Faulty	27 July, 1925	—	—
							29 Dec., 1925	Slight	Slight
18	Poor	Faulty	3 Oct., 1923	Slight	Slight	Good	30 May, 1925	—	—
							17 July, 1925		
28	Poor	Good	15 Dec., 1923	Nil	Slight	Good	31 Aug., 1925	Nil	Slight
29	Fair	Good	23 Nov., 1923	Nil	Nil	Good	31 July, 1925		
							31 Aug., 1925		
							12 Jan., 1926	Nil	Nil
40	Fair	Good	31 Mar., 1924	Nil	Nil	Good	17 July, 1925		
							6 Dec., 1925		
							29 Dec., 1925	Nil	Slight
43	Poor	Good	27 July, 1924	Slight	Slight	Good	11 Feb., 1925		
							30 May, 1925		
							27 July, 1925		
							5 Aug., 1925		
							21 Nov., 1925	Nil	Slight
47	Poor	Faulty	26 Apl., 1924	Slight	Slight	Good	17 Nov., 1925	Marked	Marked
51	Poor	Good	Before admission	Nil	Nil	Faulty	7 Jan., 1924		
							30 Apl., 1925		
							11 July, 1925		
							21 Nov., 1925	—	—
53	Fair	Faulty	9 Sept., 1924	Nil	Nil	Good	9 Feb., 1925		
							3 May, 1925		
							17 July, 1925		
							12 Jan., 1926	Nil	Slight
54	Fair	Good	28 May, 1924	Nil	Nil	Good	5 Aug., 1925		
							31 Aug., 1925		
							6 Dec., 1925		
							12 Jan., 1926	Marked	Slight

Case No.	Health.	Habits.	Inoculated.	Improvement.		Habits.	Reinoculation.	Improvement.	
				Mental.	Physical.			Mental.	Physical.
62	Poor	Good	29 Apl., 1924	Nil	Slight	Good	5 Aug., 1925		
							31 Aug., 1925		
							21 Nov., 1925	Slight	Marked
63	Bad	Faulty	29 Apl., 1924	Nil	Slight	Good	31 July, 1925	—	—
71	Bad	Faulty	6 May, 1924	Nil	Marked	Good	24 May, 1925		
							17 July, 1925		
							6 Dec., 1925		
							12 Jan., 1926	Nil	Nil
75	Fair	Good	13 May, 1924	Nil	Nil	Good	10 Feb., 1926	Nil	Nil
77	Poor	Good	2 July, 1924	Slight	Slight	Good	31 Aug., 1925	Marked	Slight
78	Bad	Faulty	27 July, 1924	Nil	Slight	Good	13 Apl., 1925	Slight	Marked
79	Bad	Faulty	24 Aug., 1924	Nil	Marked	Good	11 Feb., 1925		
							6 Dec., 1925		
							29 Dec., 1925	Marked	Slight
92	Poor	Good	15 Mar., 1925	Slight	Marked	Good	17 Nov., 1925	Nil	Nil
99	Fair	Faulty	17 July, 1925	Slight	Slight	Good	12 Mar., 1926	Nil	Nil
100	Poor	Faulty	4 Aug., 1925	Slight	Slight	Good	17 Mar., 1926	Nil	Nil
108	Fair	Good	Aug., 1924	—	—	Good	17 Nov., 1925	Slight	Nil
136	Fair	Good	14 Apl., 1924	Marked	Slight	Good	—	—	—
	Poor	Faulty	—	—	—	—	26 June, 1926	—	—

Case No. 3.—Died June, 1925.
Case No. 8.—Discharged July 1926.
Case No. 18.—Died May 1926.
Case No. 51.—Died January 1926.
Case No. 54.—Discharged August 1926.
Case No. 63.—Died December 1925.
Case No. 77.—Discharged January 1926.
Case No. 99.—Died June 1926.
Case No. 51.—This patient was treated before admission to this hospital, and details of his pyrexia are unknown, except that he had 10 rigors, and was subsequently discharged.
Case No. 136.—This patient was treated in this hospital and improved sufficiently after his first induced malaria to be discharged in February 1925. He relapsed and was re-admitted as shown in the Table. Prior to re-admission, patient had suffered severe privation, and had over-indulged in alcohol.

In all these cases a known strain of benign tertian type has been used, but thirteen instanced pyrexia of the double variety ; sixteen had a spontaneous disappearance of the parasites, while two received quinine medication after very few rigors because of intercurrent complications ; the remaining two had quinine exhibited in the ordinary way, when it was determined to terminate the attack. In contrast to this, out of a series of over 100 first attacks only five patients did not require quinine to end their fever, some of these being accounted for by the fact that they had had mosquito-acquired malaria in the tropics.

Owing to this frequent spontaneous remission we have not been able to obtain as many rigors per patient as we would have wished : whereas the average number per patient for first malaria has been seven, second attacks give us only five.

Before considering results we would mention that all these cases have been under observation on an average for eight months since their second malaria.

Of the twenty cases under review one patient has since died, and seven have shown no improvement whatsoever : seven have shown some slight improvement, either mental or physical ; two have made marked progress, especially mental, and have good prospects of an early discharge, and three have already been sent home.

It may be of interest to give fuller particulars concerning these last five cases.

CLINICAL HISTORIES.

Case 47. Admitted December 1923, age 50, health poor and habits faulty. C.S.F. Lange 5555521000, globulin markedly positive.

Inoculated in April 1924, and had 11 rigors.

He was slightly improved mentally, and his physical condition was better, while his habits were good.

In August 1925 C.S.F. Lange 0133332100, with globulin negative.

Reinoculated in November 1925, and had 7 rigors which terminated spontaneously.

His mental condition is markedly improved, and he is a useful, intelligent and reliable worker, and his initiative and insight are very fair. His bodily health is uniformly good.

In February 1926 C.S.F. Lange 0011110000, with globulin and acetic anhydride tests negative.

His discharge is now being considered.

Case 79. Admitted June 1924, age 32, bed-ridden condition and habits faulty. C.S.F. Lange 5555310000, globulin markedly positive.

Inoculated in August 1924, and had 2 rigors.

He was little, if any, improved mentally, but his physical condition was very much better, and his habits were good.

In October 1925 C.S.F. Lange 0344433000, with globulin negative.

Re-inoculated in February and early December 1925, and successfully in late December of last year. He had 5 rigors which terminated spontaneously.

His mental condition is now very much improved and although very reserved he works usefully and in a consistent manner, with a considerable interest in his late occupation and home affairs. He is rational, pleasant and appears to be stable. His discharge may be considered shortly.

In April 1926 C.S.F. Lange 1233332100, with globulin negative, and acetic anhydride test negative.

CASE 8. Admitted January 1923, age 38, health poor but with good habits. C.S.F. Lange 5555554321, globulin markedly positive. Admitted with a history of cerebral seizures.

Inoculated April 1924, and had 11 rigors.

He showed a slight mental improvement, and a marked physical one. His habits however, became slightly faulty and he had occasional repeated seizures.

In August 1925 C.S.F. Lange 4555544300, with weakly positive globulin.

Reinoculated in July 1925, and successfully in December 1925. He had 5 rigors, terminating spontaneously.

He subsequently showed sufficient improvement to be discharged a short time ago.

CASE 54. Admitted December 1923, age 35, health fair and with good habits. C.S.F. Lange 5555555410, globulin heavily positive.

Inoculated May 1924, and had 7 rigors, with two relapses subsequently.

He was improved neither mentally nor physically; in fact, his health was slightly impaired.

In August 1925 C.S.F. Lange 0155432000, with negative globulin.

Reinoculated in early and late August 1925, December 1925, and successfully in January 1926. He had 4 rigors, terminating spontaneously.

Physical improvement was noted almost immediately on the termination of pyrexia, while his mental condition steadily cleared up, and he was subsequently discharged.

CASE 77. Admitted June 1924, age 46, health poor but habits good. C.S.F. Lange 5555555543, globulin markedly positive.

Inoculated July 1924, and had 8 rigors.

There was little, if any, improvement on the mental side, although his health was a trifle more robust.

In August 1925 C.S.F. Lange 0125543300, with negative globulin.

Reinoculated August 1925, and had 4 rigors, which terminated spontaneously.

His improvement was steady and continuous, and his health became excellent. He was finally discharged and reports have been received that he is doing very well at his ordinary business.

CONCLUSION.

In view of a number of cases where there has been little or no mental improvement subsequent to the first therapeutic malaria, and yet where definite progress followed a second attack, it seems well worth while to renew malarial treatment in suitable patients.

We wish to thank our Medical Superintendent, Dr. F. M. Rodgers, for his kindness in permitting us to quote hospital records.

REFERENCES.

[1] NICOLE and STEEL, *Jour. of Trop. Med. and Hyg.*, Dec. 1925.
[2] NICOLE and STEEL, *Jour. of Trop. Med. and Hyg.*, Feb. 1926.

A CASE OF ASTASIA-ABASIA AND SPEECH PERSEVERATION, FOLLOWING CARBON MONOXIDE POISONING.

By H. G. WOLFF, New York.*

INTRODUCTION.

Edsall[1] lists forms of neuritis, palsies, anæsthesias, neuralgias, choreiform movements, intention-tremors, scanning and stuttering speech, incontinence of urine, Landry's syndrome, multiple sclerosis, blindness, and marked mental changes, as sequelæ of carbon monoxide poisoning. McGurn[2] mentions more than one hundred neurological and other symptomatological conditions, as well as two cases of a syndrome not unlike that of multiple sclerosis. Dana[3], in the early days of corpus striatum physiology, associated the lesions observed in the basal ganglia in carbon monoxide poisoning with the symptomatology of the disease, mentioning disturbances of articulation and mentality, but assigning no great motor function to the basal ganglia. More recently Hill and Semarak[4], Stewart[5], and Rugh[6], have collected a number of data on the pathological changes seen in persons dead from gas poisoning. The latter has collected a large bibliography on the subject, consisting chiefly of cases showing one or several of the above mentioned conditions, with many reports on the pathological findings in the basal ganglia. More recently Alpers[16] has reported a case with recovery, showing evidence of complex central and peripheral derangement.

The essential features of the nervous pathology, drawn chiefly from the findings of Hill and Semarak (32 cases) and Rugh (12 cases), are as follows:

1. There is a bilateral ischæmic necrosis of the lenticular nuclei, especially of the globus pallidus. This necrosis may be due to several factors. It may result from degeneration of vessel walls with thrombosis due to destruction of red cells, or as a result of the noxious gases in the blood plasma irritating the vessel wall (carbon monoxide is non-irritant, but illuminating gas may be). A more likely explanation is that in keeping with Haldane's observations. The CO in uniting with the hæmoglobin replaces oxygen; no gaseous exchange can take place. As a result the endothelium of the vessel is impaired or destroyed, with secondary thrombosis. Still another explanation is that owing to anatomical peculiarities in circulation this particular site is chosen. Unfortunately, much as this explanation pleases, there is at present lack of evidence to prove its truth.

---

* From the Bellevue Hospital, New York; service of Dr. Foster Kennedy.

2. The degree of pathological change varies from slight perivascular lesions of the globus pallidus, to gross softening, depending on the amount of gas inhaled, the duration of life after poisoning, and the presence or absence of sclerotic changes in the vessels. Cases in which the blood-vessels show a high degree of sclerosis will, in the opinion of these observers, be found to exhibit most marked changes in the parenchymatous tissues. This has an important bearing on the disease as it occurs in elderly persons or in those with a history of chronic intoxication.

3. Nerve cells possess a high degree of tolerance to the action of the gas. This is in accord with Haggard's[7] work on embryonic nerve tissue. When such tissue was exposed to atmospheres of 79 per cent. CO and 21 per cent. O, little or no disturbance in growth was noted. It must be said, however, that when as little as ·1 per cent. of illuminating gas was used death of the tissue resulted invariably. In material from a case in which death occurred twenty-seven days after exposure, Stewart noted degeneration of the spinal nerves and the vagus. Whether this was due to a direct action of the gas on nerve tissue or to the processes described above (secondary to vessel changes) it is difficult to determine. The same observer, in addition to the findings described, noted a zone of softening of the deeper layers of the cortical gray matter, punctiform hæmorrhages, and in the spinal cord a washing out of the anterior horn cells, which tended to diminish in the caudal portion of the cord.

Hill and Semarak noted œdema of the brain in about two-thirds of their cases, internal hydrocephalus in about two-thirds, a pink colour in about one-third, petechial hæmorrhages in one half, arteriosclerosis in all, and, in more than half, degrees of vascular degeneration excessive for the age of the individual. There was gross bilateral softening of the lenticular nuclei in slightly less than half, the most marked changes being observed in older persons with vascular degeneration. Of the fifty-six per cent. (18 cases) showing no gross softening, eleven patients were under forty and thirteen died within three days.

In experimentally produced carbon monoxide intoxication[15], brain œdema has actually been seen to develop and evidence of eye changes (papillœdema) has been recorded. The severe headaches of the disease are thought to be caused by this.

Vessel changes are not limited to those supplying nerve tissue (Briggs[8]).

On the neurological side, therefore, this intoxication results in symptoms arising from several levels. Cortical degeneration might well give rise to most of the disturbances of intellect, emotion, inhibition, and possibly to sphincter and pyramidal abnormalities; subcortical degeneration to tremors, disturbances of co-ordination and facial expression, choreiform[9] or athetoid activities, and possibly nystagmus; cord and spinal nerve degeneration to neuritis and lower motor neurone types of palsy[14]. The frequency of symptoms from each of the several levels is, of course, difficult to determine. Depression

is one of the commonest complaints in chronic intoxication, but this may well be the reaction to generalized tissue invasion. In severe acute intoxications depression, amnesia, dementia, and personality changes have been described[10]. Nevertheless, the most characteristic syndromes are those which supposedly have their origin in lesions of the basal ganglia.

CASE HISTORY.

The case here presented is probably one of this class. It is unusual, in as much as few of this type, with the given etiology, have been described in the literature; and it adds another observation to the interesting problem of voluntary versus involuntary motility.

An American salesman, age 46, entered the hospital complaining of difficulty in walking and speech.

Family History.—His father hanged himself during an attack of depression, and his mother died in an insane asylum after a prolonged mental illness in old age.

Previous History.—His birth and early childhood were normal. He had none of the common childhood diseases. He played well with other children and attained a fair standing in his school work. He was sociable and friendly, although somewhat inclined to stay at home and not associate as freely with those of his own age as did other members of his family. He indulged moderately in autoerotic practices until he was about seventeen, after which he sought heterosexual expression. Industrially, he was quite successful. He occupied himself as salesman the greater part of the time, and was employed for sixteen years by the firm with which he was last affiliated. At seventeen he began drinking heavily, and for the last ten years of his alcoholism he consumed at least a quart of whisky per week. At thirty-nine he had delirium tremens, and at forty-one he ceased to use alcoholic beverages.

At the age of twenty-three he developed a depression which lasted one year. This was unaccompanied by morbid somatic sensations. At no time has the patient been aware of delusions, illusions, or hallucinations (except during the attack of delirium tremens) or compulsive phenomena. He has always readily become depressed, although his depressions were usually transitory in character. He readily loses his temper, and is sensitive to the opinion of his fellows. He has never noted prolonged or short periods of excitement. It was during a fit of depression that he attempted suicide (see below).

He married for the first time when he was twenty-eight. He was divorced by his first wife at thirty-nine because of her complaints of brutal treatment, and because of his meanness in things financial. Immediately after, he married a widow with three small children and lived happily with her until three years ago. At that time domestic trouble ensued. He attributes his domestic strife to his progressive loss of sexual power. After two months of strained relations of this type, the patient developed a 'nervous breakdown,' and became greatly depressed. He claims that he saw his wife put "pills" in his coffee, and feared that she was seeking his life. He thereupon left her and shortly after attempted suicide by gas.

Present Illness.—After his exposure to gas (three years ago), he remained unconscious for 48 hours. For a month and a half he could not 'walk or move.' Sometime shortly after this he was able to sit up and move himself about in a wheel chair. Six months after his poisoning he began to walk as at present. He devised

a cane with a hook on the lower end of it, which enabled him to drag his lagging foot forward. His speech defect could not be accurately dated, but apparently came on about the same time as his difficulty in walking. Since that time he has been much hospitalized, repeatedly diagnosed as hysteria, given much suggestive and manipulative treatment, but always presenting essentially the same picture as at present. His sister, who has nursed him, has noted, however, a gradually progressive mental change. He has become more and more untidy about his clothes, often goes about with his trousers unbuttoned, occasionally soils himself when in bed, chiefly with urine, but on several occasions, even in public places, with fæces. He eats with little regard for table manners, drops food about himself, and soils his linen regardless of reprimand. This is very different from his behaviour before the poisoning.

Physical Examination.—The patient is big and somewhat obese, and appears older than his actual age. His facies is sad or apprehensive, and immobile except during occasional outbursts of weeping. Usually there is little facial expression.

The muscles supplied by the cranial nerves show no defect, with the exception of the Parkinsonian facies. The tongue is protruded in the midline promptly and presents no atrophy or tremor. There is occasional very slight drooling of saliva. The voice is thin, high-pitched, and lacking in volume. It is also entirely lacking in intonation, and is dull and frequently explosive. Certain consonants are especially difficult to articulate. During conversation words are repeated over and over again. There is a gush of words when an answer is given to a question. For example: "How do you feel?" "I feel fine, fine, fine." It was noted that he repeatedly had difficulty in the expression of those words that had an emotional association. The rate of speech is extremely rapid. When asked to repeat slowly certain test words after the examiner, there is no perseveration, and the quality of the voice seems better, but when asked to repeat the same words without the model sounds to copy from, the perseveration reappears. Frequently when a word persistently repeats itself, he will spell the word out, as, "What colour is this chair?" "White, white, white, white . . . W H I T E," all at a very rapid pace, the spelling as rapidly as the remainder of the flow.

Motor System.—The patient stands erect. There is no muscular weakness. There is a cog-wheel rigidity at the elbow, when the forearms are passively moved. The gait is clumsy and spastic. In starting to walk, the left leg is pushed in front and the right remains behind. Thus he may remain for several moments, after which the hindmost leg may be brought slightly forward, but not in front of the foremost leg. The latter leg may then be raised slightly and pushed forward. The steps are very short and the patient alternates between shuffling and sticking. There is always especial difficulty in getting started, after which motion is continued as described. He uses a cane, and when this is taken from him he waves his arms about and leans on or towards anything that will afford support. When this is not possible, he spreads his legs and extends his arms literally in an attempt to balance himself. When the patient sits down on a chair, there is no gradual recline or 'easing' into the chair, but a descent en masse, so that he strikes with a thud and rolls well back. At times, especially in the morning, the patient walks apparently normally. The steps are still short, but the natural progression is followed. Even when his walking is at its worst, usually late in the evening or night, he is still able to step over obstacles placed in his path. There is a slight tremor of the hands. The handwriting is more or less characteristic of that commonly seen in paralysis agitans (see fig. 1). On one occasion, while addressing a letter, the patient was seen to write the name of the city twice. He plays the piano with

a peculiar rapidity, and with faulty fingering and expression, and insists that he perseverates on certain phrases, but the playing was so poor that the observer could not be certain of the fact. His playing, according to friends, is said to have materially altered, but this remains uncertain.

The retinæ show a moderate degree of arteriosclerosis, with some vessel tortuosity, but no hæmorrhages. The radials are rigid and slightly tortuous.

No abnormalities are found in the reflexes except a reduction of both abdominals and cremasterics. Tests for co-ordination are well performed. When lying in bed, the patient can touch accurately with his feet an object held in mid-air, and perform movements requiring first the one leg and then the other. Immediately after such a performance, when tested in walking, the characteristic gait was observed. No changes in sensation, either deep or superficial, are noted, and no disorder referable to the sympathetic system. The right leg is smaller in diameter than the left.

Mental State.—The patient is listless and inactive. He sits about reading newspapers or dozing in his chair. The above-mentioned untidiness in habits was noted. On one occasion the patient defæcated on the floor of the ward. He explained his action by saying that he could not get to the lavatory promptly enough. Outbursts of weeping without adequate environmental stimuli were noted on several occasions. Mild depression is the usual affect, and a peculiar childishness as regards the creature comforts (gluttonous appetite, behaviour at table, greediness, etc.) has been observed. The patient can whistle, but a true smile has never been noticed. His judgment is poor, although he expresses a constant desire to get well and return to work.

The routine laboratory studies were negative.

COMMENT.

The exact neurological classification of this syndrome is unimportant. There were periods of personality derangement, as revealed in the record (manic-depressive episodes). The history of excessive alcoholism over a long period, together with the appearance of senility, make it more than likely that the patient has a reasonable degree of cerebral arteriosclerosis. This would make the action of the gas more drastic, if the previously mentioned observation be correct.

Foix[11] calls this syndrome pseudobulbar palsy. Others prefer to limit the term, not permitting inclusion of a case such as the above. In view of the poor family and past history, some might doubt its organic nature. Oppenheim[13] mentions that he has seen a case of astasia-abasia following carbon monoxide poisoning. The suggestive facies, the cog-wheel rigidity of the musculature, the manner of sitting down, and the handwriting, leave in my mind no doubt as to the organic nature of at least part of the syndrome. Speech difficulties have been noted before[3]. The personality change and sphincter disturbance may well be ascribed to cerebral degeneration. The astasia-abasia, however, remains a difficult symptom to explain.

Assuming a segmental arrangement in the basal ganglia, as has been frequently suggested, it is conceivable the difficulty in walking may be a manifestation of a pathological change of co-ordinating mechanisms at the leg

12/19/23.

Dr Wolf.
Bellevue Hospital
New York, N.Y.

Dear Doctor:

I am about to tell you that I am going to get well & hope that with the assistance of your goodself that my hopes will be realized very soon for a speedy recovery.

There seems to be a general feeling of optimism of the patients under your care and I trust that the results you are striving for will be realized and that you will enjoy good health yourself and also that your family are all well. Am with my very sincerest regards and best wishes I am.

Yours sincerely,
Frank Sheehan
New York City
N.Y.

Fig. 1

level. Further possibilities are that the will to walk is very weak, or the resistance to be overcome in executing the act extremely great.

Kennedy, Davis and Hyslop[12] recently described vegetative changes occurring as postencephalitic symptoms, suggesting that this disease may be a factor in supplying the missing link between so-called functional and organic diseases. That volition plays a part in the symptoms of lesions of the basal ganglia is probable. Patients with severe Parkinsonian paralysis have been observed to have many variations in their clinical pictures, depending on such factors as the time of day and the emotional stress appended to an act of the will.

Foix[11], in his description of the autopsy findings in individuals with clinical pictures similar to the above, apparently agrees with most authors in stating that the characteristic syndrome must be the result of a bilateral lesion affecting association tracts on both sides, or of a unilateral lesion so placed as to intercept tracts on either side. Several types have been described, but the essential features are degeneration of the basal ganglia, some supraganglial and peduncular degeneration, and possibly changes in pons and medulla.

This case throws no light on the anatomical site responsible for disturbances in motion and behaviour. Most destructive processes in the brain due to gas are too diffuse to permit of fine discriminations and assignment of physiological activity to a definite area. I believe, however, that the case is of interest in suggesting that conditions commonly associated with psychogenic disturbances or other evidences of personality derangement may be expressions of an actual lesion in the brain or brainstem. At present we know little about what may be in common between the types of change that can be produced by suggestion on the one hand, and by severe arterial damage on the other.

REFERENCES.

[1] Edsall, *Osler's System of Medicine*, 1915, ii, 388.

[2] McGurn, *Med. Rec.*, 1917, xci, 149.

[3] Dana, C., *Jour. Nerv. Ment. Dis.*, 1908, xxxv, 65.

[4] Hill and Semarak, *Jour. Amer. Med. Assoc.*, 1918, lxxi, 644.

[5] Stewart, *Jour. Neurol. and Psychopath.*, 1920, i, 105.

[6] Rugh, *Arch. f. Psychiat.*, 1921, lxiv, 150 (Complete bibliography up to 1921).

[7] Haggard, *Amer. Jour. of Physiol.*, 1922, ix, 244.

[8] Briggs, *Jour. Amer. Med. Assoc.*, 1919, lxxiii, 678.

[9] Merguet, *Arch. f. Psychiat.*, 1922, 583.

[10] Hitchcock, C. W., *Jour. Amer. Med. Assoc.*, 1918, lxxi, 257.

[11] Foix, *Nelson's Loose Leaf Med.*, 1920, vi, 461.

[12] Kennedy, Davis and Hyslop, *Arch. Neurol. and Psychiat.*, 1922, viii, 40.

[13] Oppenheim, *Text Book of Nervous Diseases (English Ed.)*, 1911, ii, 1159.

[14] Wilson and Winkelman, *Jour. Amer. Med. Assoc.*, 1924, lxxxii, 1407.

[15] Forbes, H. S., Cobb, S. and Fremont-Smith, F., *Arch. Neurol. and Psychiat.*, 1924, xi, 264.

[16] Alpers, B. J., *Amer. Jour. Med. Sci.*, 1925, clxx, 390.

THE ACETIC ANHYDRIDE TEST IN CEREBROSPINAL FLUID.

By J. G. GREENFIELD and
E. ARNOLD CARMICHAEL, London.*

The object of this paper is to place on record the results of the sulphuric acetic anhydride test in a number of cerebrospinal fluids, and further to discuss the possible underlying 'causal factor.'

The work has been done in the Pathological Laboratory of the National Hospital, Queen Square, and we desire here to tender our thanks to the physicians of the hospital for permission to publish these reports. In all, the series includes 50 cases ranging from non-organic conditions of the central nervous system to dementia paralytica.

LITERATURE.

In 1923 Boltz[1] published a paper on "Studies of the Cerebrospinal Fluid with an Acetic-anhydride Sulphuric Acid Test." This is, so far as we have been able to ascertain, the first paper upon this subject. In examining a large series of cases he found that the reaction gave a positive finding only in cases of dementia paralytica and taboparesis. He had the opportunity of examining 22 fluids from cases of dementia paralytica, and in every one found a positive result; in two cases of taboparesis, one gave a positive, the other a slight positive. No other positive reactions were obtained. Discussing the 'causal factor,' he states that the substance is not destroyed by heating the cerebrospinal fluid, and assumes therefore that it cannot be analogous to the substance giving the colloidal gold reaction. Further, he found that the 'causal factor' was thrown down with the globulin in Noguchi's test, as after so doing the supernatant fluid gave a negative result, while a saline solution of the centrifuged deposit gave a positive result. He lastly argued that though the test is similar to a cholesterol reaction its positivity in such cases "cannot be due to excess of cholesterol, as Weston states that the cholesterol content in the cerebrospinal fluid of dementia paralytica is not as high as that in cases of other forms of cerebral degeneration."

Grossman[2], in 1925, found the test to be positive in 28 cases of general paralysis in a series of tests upon 60 cases. The remainder of the cases, all of which gave negative results, were those of patients suffering from psychoses, epilepsy and other non-syphilitic conditions. In opposition to Boltz he states that the reaction is dependent upon the cholesterol content of the spinal fluid.

* From the Laboratory of the National Hospital, Queen Square, London.

It is closely similar to the reactions of Salkowski and Liebermann-Burchard, by which the cholesterol content of various body fluids is determined.

Harris[3] in the early part of the present year recorded a further series of cases to which this test had been applied. He, too, found that it was specific for general paralytic fluids. In 92 cases of general paralysis he found this test positive in 84 (91 per cent.); five cases of neurosyphilis were examined, two of which gave a positive result, and from 83 cases of other types of mental disorder he obtained only one positive. He made a further interesting observation that malarial treatment, though changing the strength of the Wassermann reaction, did not affect the acetic anhydride test. From his observations he concluded that in general paralysis this test was almost as reliable as the Wassermann reaction, and he suggested the possibility of the reaction depending upon the presence of cholesterol in the cerebrospinal fluid.

Steel and Nicole[4] have published their results of this reaction upon fluids of a large series of general paralytics and non-syphilitic patients. They have only recorded the results of three reactions, the Lange, the globulin, and the acetic anhydride. In 39 fluids from non-syphilitic patients no positive reaction with acetic anhydride was obtained. In 36 fluids from paretic patients they only obtained one negative result; the globulin reaction was also negative in this isolated case. In eight fluids out of a total of 30 from patients suffering from general paralysis who had had malarial treatment the result of the acetic anhydride test was negative. They state that this reaction is more sensitive than the globulin reaction, and remains unaltered by malarial treatment for a longer period than the globulin or colloidal gold reaction.

TECHNIQUE.

We have used the following technique in performing this test. The cerebrospinal fluid used was never over 48 hours old and had not been heated; if the test could not be performed at once the fluids were kept in an ice chest. Our experience has shown us that fluids which give a positive result may be kept for a week or more in the ice-chest without any change in the result of the test when repeated.

One c.c. of the cerebrospinal fluid is measured into a test-tube with a graduated pipette. To this is added 0·3 c.c. of *chemically pure* acetic anhydride. The test-tube is then gently agitated to cause an emulsion to form. To this is then added drop by drop 0·8 c.c. of chemically pure sulphuric acid. Should no colour immediately appear, the test-tube is again shaken gently when invariably some colouration takes place.

Our experience has shown us that in almost every instance a change in colour begins to appear after 0.6 c.c. of sulphuric acid has been added and that the consequent addition of a further 0·2 c.c. has merely intensified the original colour. Further, we have observed that unless a reading be immediately taken after the addition of the sulphuric acid further colour changes may take

place and give a wrong result. In other words, the reaction is a rapid and fleeting one, and requires immediate recording.

A positive result is constituted by a very definite lilac colour—it may range from a comparatively light to a deep lilac. Other colours such as pink, blue-pink or brown were considered negative. In fact, any colour other than a definite lilac must be considered without dubiety as negative. Our experience has led us to associate a dirty brown colour with a high protein content.

In our series we have also made a routine examination of all the fluids. Our records thus not only include the results of the acetic anhydride test but also the physical characters of the fluid, the cell content, the total protein and the Wassermann reaction in the spinal fluid of all and in the blood of most of the cases. We are thus in a position to discuss whether a positive acetic anhydride test is constantly associated with a positive result from one of the other reactions or whether it may be found only in cases which show a positive result in all reactions; or whether it may be due to some 'causal factor' so far not found to influence the usual routine reactions, the causation of which is known.

RESULTS.

Our results are given in the table. The diagnosis of each case has been made upon clinical grounds and is not the result of biochemical and biological criteria.

G.P.I. FLUIDS

	Sex.	*Acetic Anhydride Test.*	*Physical Characters.*	*Cells.*	*Total Protein.*	*Nonne-Apelt.*	*Pandy*	*Lange.*	*C.S.F. W.R.*	*Blood W.R.*
E.B.	M.	Deep lilac.	Clear, colourless.	32· 10% large mono. 15% poly.	0·1%	pos. +++	pos. +++	2344533221	4444	4443
G.S.	M.	Deep lilac.	Clear, colourless.	45·0 25% large mono.	0·075%	pos. ++	pos. ++	3533432100	4444	44??
A.P.	M.	Lilac	Slightly yellow.	40·0 10% large mono.	0·1%	pos. +++	pos. +++	5555555543	4444	4210
W.	M.	Lilac	Clear, colourless.	14·0	0·06%	pos.	neg.	3321000000	4444	4444
R.	M.	Deep lilac.	Clear, colourless.	40·0 5% large mono.	0·045%	pos. +	pos. +	4555554443	4444	4444
G.B.	M.	Brown lilac.	Clear, colourless.	15·0 15% large mono.	0·08%	pos. ++	pos. ++	5555555432	4440	neg.

	Sex.	*Acetic Anhydride Test.*	*Physical Characters.*	*Cells.*	*Total Protein.*	*Nonne-Apelt.*	*Pandy*	*Lange.*	*C.S.F. W.R.*	*Blood W.R.*
L.I.	M.	Lilac	Clear, colourless.	6·0	0·05%	pos. +++	pos. +++	3200455555	4444	4443
W.R.	M.	Lilac	Clear, colourless.	82·0 5% large mono.	0·1%	pos. +++	pos. +++	3445555432	4444	—
H.P.	M.	Lilac	Clear, colourless.	7·0	0·065%	pos. ++	pos. ++	4555555533	4442	4443
A.L.	M	Lilac	Clear, colourless.	60·0 10% large mono.	0·10%	pos. ++	pos. ++	3555444443	4444	4443
R.F.	M.	Lilac	Clear, colourless.	36·0	0·09%	pos.	pos.	5555555553	4444	3200
S.	M.	Lilac	Clear, colourless.	8·5 5% large mono.	0·06%	pos. ++	pos. ++	2444555543	4443	4444
B.	F.	Lilac	Clear, colourless.	14·0 4% large mono.	0·045%	weak pos.	weak pos.	0012100000	1000	—
S.	M.	Lilac	Yellow	57·0 5% large mono.	0·09%	pos. ++	pos. +++	4555554332	4442	—
F.	M.	Lilac	Clear, colourless.	45·0 5% large mono.	0·06%	pos. ++	pos. ++	5555554211	4444	4443
M.	F.	Lilac	Clear, colourless.	75·0 15% large mono.	0·075%	pos.	pos. ++	3444333221	4444	4443
				FLUIDS OF TABES DORSALIS.						
H.	M.	Pink-blue.	Clear, colourless.	1·0	0·45%	very weak pos.	pos. +	No change	neg.	neg.
J.	F.	Yellow	Clear, colourless.	19·0	0·03%	weak pos.	pos.	2233443322	neg.	pos.
M.	M.	Pink	Clear, colourless.	45·0 10% large mono.	0·1%	pos. ++	pos. ++	0232221110	4444	4444
S.	M.	Lilac pink.	Clear, colourless.	1·0	0·04%	neg.	neg.	0001110000	neg.	neg.

	Sex.	*Acetic Anhydride Test.*	*Physical Characters.*	*Cells.*	*Total Protein.*	*Nonne-Apelt.*	*Pandy*	*Lange.*	*C.S.F. W.R.*	*Blood W.R.*
G.	F.	Light pink.	Clear, colourless.	3·0	0·03%	neg.	neg.	0001101000	neg.	neg.
S.	M.	Yellow	Clear, colourless.	52·0 8% large mono.	0·04%	neg.	neg.	0011000000	4200	4442
				FLUIDS OF OTHER FORMS OF SYPHILIS.						
S.J.	M.	Lilac	Yellowish turbid.	85·0 10% poly, 5% large mono	0·8%	pos. +++	pos. +++	0000011112	4442	4433
F.	M.	Bright pink.	Clear, colourless.	36·0	0·09%	pos.	pos.	5555555553	4444	3200
R.	F.	Light lilac-pink.	Clear, colourless.	56·0 5% large mono.	0·045%	neg.	weak pos	0000100000	4421	?
B.	M.	Light lilac-pink.	Clear, colourless.	20·0	0·03%	neg.	very weak pos.	4444332100	4443	4433
				OTHER CONDITIONS.						
TUMOURS OF BRAIN AND CORD.										
G.	F.	Brown	Clear, colourless.	?	0·02%	neg.	neg.	—	neg.	neg.
J.	M.	Pink	Slight yellow.	?	0·03%	neg.	neg.	1122200000	neg.	neg.
L.	F.	Pink	Clear, colour less.	1·0	0·15%	pos. +	pos. +	0001122110	neg.	neg.
Q.	M	Brown	Clear, colourless.	0	0·03%	weak pos.	weak pos.	0002333000	neg.	neg.
L.	M.	Pink	Clear, colourless.	0	0·065%	weak pos.	weak pos.	—	?	?
S.*	F.	Brown	Slightly yellow.	2·0	0·09%	pos. +	pos. +	No change	neg.	neg.
C.	M.	Pink	Clear, colourless.	3·0	0·1%	pos.	pos.	0000012221	neg.	neg
M.	M.	Brown	Yellow & coagul'm	5·5	0·8%	pos.	pos.	0000111233	neg.	neg.

	Sex.	*Acetic Anhydride Test.*	*Physical Characters.*	*Cells.*	*Total Protein.*	*Nonne Apelt..*	*Pandy*	*Lange.*	*C.S.F. W.R.*	*Blood W.R.*
SUBACUTE COMBINED DEGENERATION.										
B.	F.	Pink	Clear, colourless.	0	0·03%	neg.	very weak pos.	No change	neg	neg.
T.	F.	Brown	Clear, colourless	0	0·025%	neg.	neg.	3444200000	neg.	neg.
LEPRA NEURITIS.										
D.	F.	Brown	Clear, colourless.	3·0	0·06%	weak pos.	pos.	No change	neg.	neg.
ENCEPHALITIS LETHARGICA (CHRONIC).										
W.	M.	Pink	Clear, colourless.	9·0	0·04%	neg.	neg.	—	neg.	neg.
VASCULAR.										
K.	M.	Brown	Clear, colourless.	0	0·025%	neg.	neg.	No change	neg.	neg.
DISSEMINATED SCLEROSIS.										
S.	F.	Yellow	Clear, colourless.	3·0	0·02%	neg.	weak pos.	No change	neg.	neg.
K	F.	Pink	Clear, colourless	4·0.	0·03%	weak pos.	weak pos.	0112110000	1000	1000
W.	F.	Pink	Clear, colourless.	20·0 10% mono.	0·0356	pos. +	pos. +	5454331100	neg.	neg.
MENINGITIS										
L.	M.	Pink	Yellow and coagul'm	++ poly	1·0%	pos. +++	pos. +++	—	—	—
CEREBRO-MACULAR DEGENERATION.										
C.	F.	Light lilac	Clear, colourless.	1·0	0·02%	neg.	neg.	No change	neg.	neg.
FLUID OF NON-ORGANIC CASES.										
G.	M.	Light pink lilac.	Clear, colourless.	4·0 1 endothelial.	0·04%	neg.	weak. pos.	No change	neg.	neg.
B.	F.	Light pink.	Clear, colourless.	3·0	0·025%	neg.	neg.	No change	neg	neg.
M.	M.	Light pink.	Clear, colourless.	1·0	0·02%	neg.	neg.	—	neg.	neg.
B.	F.	Brown pink.	Clear, colourless.	0	0·025%	neg.	neg.	—	neg.	neg.
S.	M.	Brown pink.	Clear, colourless.	4·0	0·025%	neg.	neg.	No change	neg.	neg.
H.	M.	Brown pink.	Clear, colourless.	0	0·025%	neg.	neg.	—	neg.	—

Of 16 cases of dementia paralytica the acetic anhydride test was positive in 15, the remaining fluid gave a dirty brown-blue colour, which was considered negative. On examination of the table it will be seen that except for one of these cases the fluids were all clear and colourless; that the cell counts varied from 82 to 6 per cmm., one fluid containing a few polymorphs; that the total protein varied from 0·1 to 0·045 per cent.; that the Nonne-Apelt reaction was positive (+ to +++) in all; that the Pandy reaction was positive in all except one; that the Lange gold-sol curves were all of the paretic type except two; that the Wassermann reaction was positive in all the fluids and in all but one of the bloods examined. It is therefore manifest that all the cases examined gave a typical cerebrospinal fluid picture of dementia paralytica.

Cases of tabes dorsalis gave various colour reactions varying from yellow to lilac pink; but no fluid exhibited an undoubted general paralysis reaction. A case of secondary neurosyphilis gave a definite lilac colour. Other forms of syphilis, as exemplified by cerebral syphilis, gave negative results.

The only other fluid with a positive result was from a patient suffering from familial cerebro-macular disease. The intensity of the lilac colour was very weak.

It is of interest to look at the results in the other cases as, on examining the table, reports will be found upon fluids which showed isolated abnormalities. For instance, a fluid from a case of brain tumour, yellow in colour, gave a negative result. Again, several fluids containing a marked increase of the total protein content, as high as 1·0 per cent., failed to give a positive acetic anhydride reaction. It will also be noticed that positive Nonne-Apelt and Pandy reactions are possible with a negative reaction. Lastly, it is evident that changes in the Lange reaction are not associated with the reaction under discussion.

CONCLUSIONS.

The examination of this series of fluids clearly shows that the reaction cannot be associated with any single one of the usual syphilitic reactions, or with any known combination of these. It is possible to get a negative acetic anhydride reaction where there is an increase in cells and globulin, a paretic type of Lange curve, and a positive Wassermann reaction. Accordingly, it is only fair to assume that the 'causal factor' of the reaction must be some other substance in the fluid than that producing the other reactions. The suggestion that it may be due to an increase in the cholesterol content of the fluid appears a possible explanation.

We have found that a positive reaction occurs in practically every case of dementia paralytica, only one proving negative in our series; that a positive reaction may occur in secondary neurosyphilis; and that cases of cerebral degeneration may give a weak reaction.

REFERENCES.

[1] Boltz, *Amer. Jour. Psychiat.*, 1923, iii, 111.
[2] Grossman, *Jour. of Ment. Sci.*, 1925, lxxi, 439.
[3] Harris, *Brit. Med. Jour.*, 1926, i, 136.
[4] Steel and Nicole, *Brit. Jour. Ven. Dis.*, 1926, ii, 243.

THE MANNER OF INVASION AND DESTRUCTION OF BRAIN TISSUE BY SPONGIOBLASTOMA.

By JOSHUA H. LEINER and
WALTER M. KRAUS, New York.*

In July 1918 Strauss and Globus,[1] and shortly thereafter Ribbert,[2] reported a type of cerebral neoplasm which grew very rapidly, which was made up of undifferentiated glia cells, and which was termed by them 'spongioblastoma.'

In August 1925, the former writers[3] reported a new group of sixteen cases showing the same embryonal undifferentiated glia pictures, which they now called 'spongioblastoma multiforme' in distinction to the 'medulloblastomas' of Bailey and Cushing.[4]

The present case also began acutely, progressed very rapidly to a fatal termination (within fifty-three days) and at autopsy revealed a spongioblastoma, the size of a hen's egg. Histopathologically, this single case shows all the pictures previously described, i.e., giant cells, rosette-formation, glandular appearance with radial arrangement of cells and circular cell arrangement round blood vessels.

The body of the tumour is highly cellular, very vascular and in the main of ectodermal character. The cells are either pyriform or round and show little cytoplasm; the nuclei are well stained and contain numerous coarse granules. Elongated cells are also present, as well as numerous giant cells containing many nuclei peripherally placed. We wish to lay stress on the manner of invasion of normal tissue and the formation of rosettes.

The tumour advances in a pseudopodium-like manner (Fig. 1), engulfing and then completely surrounding areas of normal brain tissue as well as blood vessels. In advance of the invading tumour there are numerous areas of colloid degeneration (Figs. 1 and 2). From the sides of the pseudopodia offshoots are seen. The cells in the sides of the invading pseudopodia are arranged in parallel fashion (Fig. 3). When viewed closely, there are areas where the most advanced part of a pseudopodium has joined a neighbouring similar part and has formed an alveolar gland-like structure, which still retains the parallel cell arrangement. It is in this way that a rosette is formed. Another manner of rosette-formation is easily understood by imagining a

* Read at the Section of Neurology and Psychiatry, New York Academy of Medicine, February 9th, 1926. From the Medical Service of Lebanon Hospital and the Laboratory of Neuropathology, Montefiore Hospital, New York City.

pseudopodial offshoot cut in a plane horizontal to that seen in Fig. 1. Since the cells are polarized towards the normal tissue and also towards the core of the offshoot, rosette-formation must occur on cutting it across near its apex. Later, glia fibres are seen to arise and converge towards the centre, which then breaks down and finally gives rise to an area of degeneration (Fig. 4).

The cells in the pseudopodium-like advancing tumour are always polarized in the direction of growth, i.e., their longest diameter points in this direction (Fig. 3). This polarization recalls that seen in embryonic connective tissue. The forces producing it are dependent, we believe, upon a distinct physico-chemical difference between the normal tissue invaded and the tumour tissue itself. This polarization and resulting parallel arrangement of the cells, combined with the pseudopodium-like advance, engulfing normal tissue, are, as suggested in less detail above, the origin of rosette-formation. Neurobiotactic processes are therefore responsible for the typical pictures of spongioblastomata (Figs. 5 and 6).

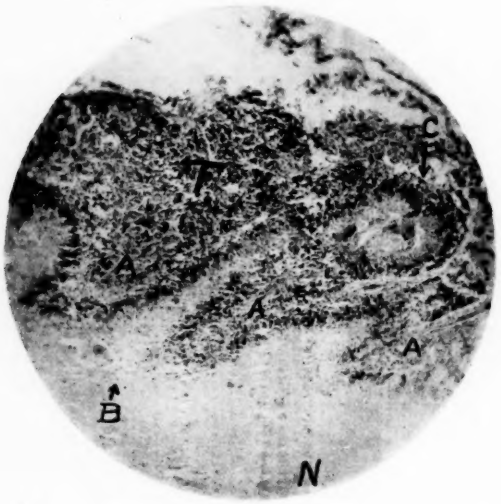

FIG. 1.—Showing the pseudopodium-like invasion of normal tissue by the tumour, and the colloid degeneration within the normal tissue in advance of the tumour. (*See also* Fig. 2.) As these pseudopodia close in on each other rosettes are formed. Hæmatoxylin-eosin. Leitz obj. I, oc. I. A, pseudopodia; B, colloid degeneration; C, rosette formation; N, normal brain tissue; T, tumour.

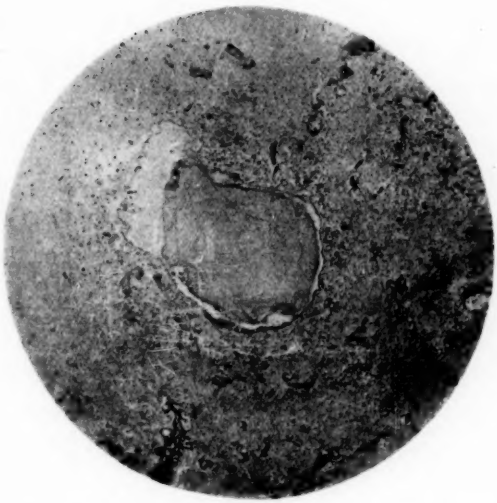

FIG. 2.—Colloid degeneration in normal tissue in advance of the invading tumour. Hæmatoxylin-eosin. Zeiss obj. 16 mm. ap., oc. 10 x comp.

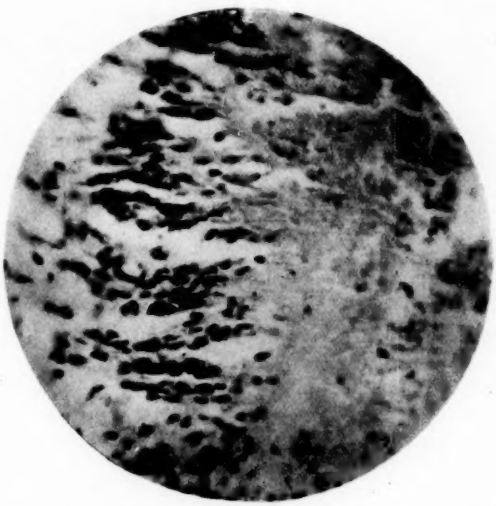

FIG. 3.—Showing parallel arrangement of the cells of the advancing tumour in the periphery of the pseudopodia. Hæmatoxylin-eosin. Zeiss obj. 4 mm. ap., oc. 10 x comp.

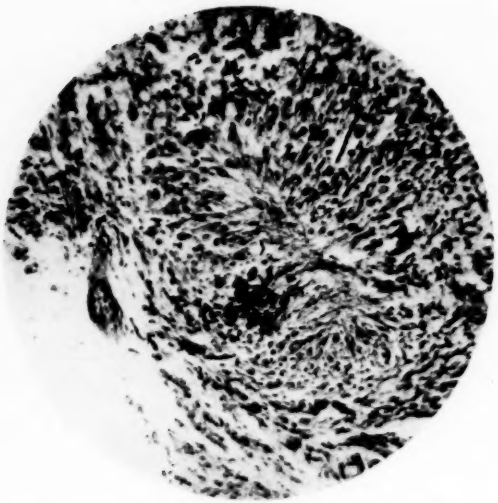

FIG. 4.—Rosette with glia fibres converging towards the centre of the core. Rio-Hortega stain, 1st modification. Zeiss obj. 16 mm. ap., oc. 15 x comp.

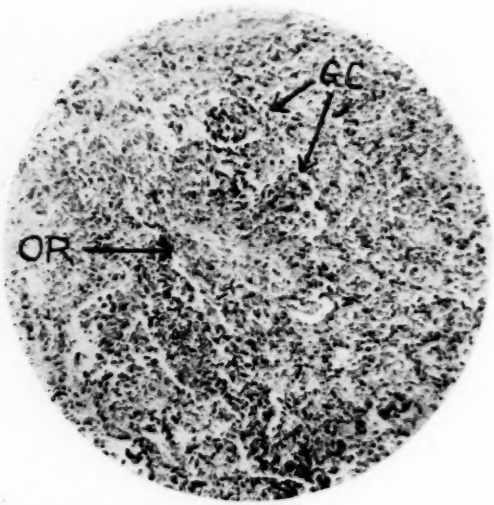

FIG. 5.—General appearance of tumour. O.R., old rosette formation; G. C., giant cells (*See also* FIG. 6). Hæmatoxylin-eosin. Zeiss obj. 16 mm. ap., oc. 7 x comp.

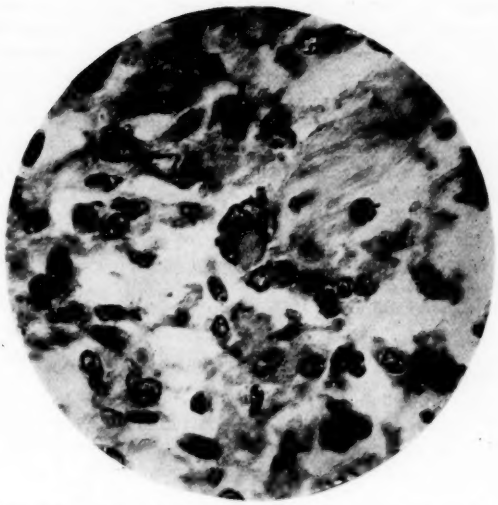

FIG. 6.—Giant cells. Hæmatoxylin-eosin. Zeiss obj 4 mm. ap., oc. 20 x comp.

CASE REPORT.

U. D., male, age 43, iron-worker, admitted to Lebanon Hospital, July 5, 1924, and died July 22, 1924. Chief complaints: (1) frontal headaches; (2) noise in head and ears; (3) weakness in both legs for the past three weeks; (4) stiffness of neck. The patient enjoyed good health until four weeks before admission, when he began to suffer from attacks of dizziness and faintness, accompanied by a severe burning sensation in the region of the left triceps. These attacks came on every morning and continued for two weeks. Headaches at this time were accompanied by a loud roaring sound, followed by vomiting. Stiffness of the neck and progressive weakness of both legs compelled him to take to bed.

Neurological Examination: July 9, 1924.

Cranial Nerves: (I) bilateral anosmia. (II) a beginning bilateral papilloedema. (III), (IV), (VI) pupils widely dilated (atropin). Weakness of left levator palpebræ; left palpebral fissure wider than right. Slight weakness of left abducens. Nystagmoid movements on right lateral fixation. (VII) a central left facial paresis. (VIII) hearing diminished to watch and tuning fork bilaterally. Bone conduction affected on left. (XII) tongue slightly deviated to left.

Movement and Co-ordination: Motor power diminished in left upper extremity, including grasp. Left lower extremity, motor power affected. Slight paresis, right upper and lower extremities. Station and gait: no Romberg, no staggering, but head tilted to left. Slight ataxia in finger-nose test on left. No spontaneous past-pointing.

Sensation: Repeated sensory examinations were of little value, because of non-cooperation on the part of the patient, but it was thought there was some hypæsthesia and hypalgesia over the trunk and both lower extremities.

Reflexes: Deep reflexes moderately exaggerated in left upper and lower extremities, but distinct diminution of left triceps jerk. Abdominals and cremasterics absent on left. An inconstant Oppenheim reflex present bilaterally.

Summary: A definite left pyramidal tract involvement, some implication of right side, and a beginning bilateral choked disc.

Course : Repeated neurological examinations showed changing phenomena. On July 15, 1924, a definite bilateral papillœdema of three to four diopters was present. Left facial paresis cleared up entirely. Knee jerks and ankle jerks present and normal in response. A right Oppenheim reflex was now elicited, but no Babinski response or ankle clonus. Abdominals and cremasterics present. A constant absence of the left triceps jerk was an outstanding feature. Sensory examination, performed repeatedly, also varied, but a persistent belt of hyperæsthesia from the third to the sixth cervical segments was present. Rigidity of the neck and a bilateral Kernig sign were found, as well as a left Babinski response and right Oppenheim reflex. The definite belt of hypæresthesia from C3 to C6 persisted.

An X-ray of this region showed slight haziness of the spinous processes. A laminectomy from the second to the seventh cervical segment revealed an œdematous dura with spinal fluid under increased pressure. A probe passed up and down encountered no obstruction. (Dr. L. Miller Kahn of Lebanon Hospital.)

Caloric examination was difficult as the patient did not cooperate well. However, it was shown that responses in the horizontal positions were normal. On July 23, he became very drowsy and feeble. A subtemporal decompression was done, but he died a few hours after the operation was terminated.

Necropsy : Limited to brain only. There was some increase in the size of the ventricles, and hæmorrhage at the sight of decompression. Incision revealed a subcortical tumour, brownish tinged in colour, the size of a hen's egg, situated in the right temporoparietal region. It was firmer in consistency than the surrounding brain tissue, and apparently encapsulated.

REFERENCES.

[1] STRAUSS, I., and GLOBUS, J. H., "Spongioblastoma with unusually rapid growth following decompression," *Neurol. Bull.*, 1918, i, 273.

[2] RIBBERT, H., "Ueber das Spongioblastoma und das Gliom," *Virch. Arch.*, 1918, ccxxv, 1925.

[3] GLOBUS, H. J., and STRAUSS, I., "Spongioblastoma multiforme," *Arch. of Neurol. and Psychiat.*, 1925, xiv, 139.

[4] BAILEY, P., and CUSHING, H., "Medulloblastoma cerebelli," *Arch. of Neurol. and Psychiat.*, 1925, xiv, 192.

Short Notes and Clinical Cases.

A CASE OF MENINGISMUS OF OBSCURE ORIGIN FOLLOWING STRETCHING OF THE ADDUCTORS OF THE HIPS.

By M. F. FORRESTER BROWN and R. G. GORDON, Bath.

The following case is published in the hope that a discussion may be provoked which may throw light on the causation, as no similar case could be traced in the literature, and none of the causes of meningismus referred to in text-books and articles on the subject were found operative in this one.

Our attention was first drawn to the possible association of meningismus with mechanical stretching of the hip adductors by a case of tubercular disease of the hip in a girl of ten. After the hip, the condition of which was quiescent, had been stretched on a frame to obtain further corrective abduction, she developed a temperature with meningeal symptoms of headache, stupor and neck rigidity. These all passed off in 36 hours without lumbar puncture, after the child was removed from the frame and the tension relaxed. However, as the gastro-intestinal functions were deranged at the time, it was supposed that the condition was toxæmic, either from absorption from the bowel, or possibly from the tubercular focus.

The present case, which is much more definite, is that of a girl, age nine years, admitted to hospital for the treatment of neglected congenital dislocation of the hips, who, shortly after the application of a Jones' hip-abduction frame, developed signs of meningismus, with transient loss of consciousness, slight convulsions and great increase of tension in the cerebro-spinal fluid, which, however, was clear and sterile on culture. Removal of the frame seemed to play a part in the clearing up of the symptoms, although some weeks later it was re-applied without actual recurrence; nevertheless, every time the tension on the hip adductors was increased by abducting the leg-pieces one or two holes, there was a small rise of temperature the same night.

The problems are: Can the cerebral symptoms have been induced entirely by mechanical means, possibly through some obscure reflex disturbance of vascular tonus under the control of the vegetative nervous system, or was the connection quite a chance one, in which latter case, what was the cause of the meningismus?

It is obvious that, if muscle stretching alone can be proved to have acted as an exciting cause, the case is of great interest to all those who have under their care large numbers of orthopædic cases, requiring treatment by this, or similar, apparatus.

The following are the details of the case.

CASE HISTORY.

Stella H., age nine years, with no history of previous illnesses, except a fall from a table at one year, was admitted to Children's Orthopædic Hospital, Bath, on August 8, 1926, suffering from unreduced congenital dislocation of both hips. She had extreme lordosis and walked badly with a waddling gait, the hips being very unstable.

Aug. 9. Abduction frame applied, practically no tension being put on adductors of the hips.

Aug. 16. Tooth extracted under ether, there being nothing unusual to note in connection with the anæsthetic.

Aug. 27. A small deep boil which had been present on one buttock since admission, increased in size and was fomented.

Aug. 28. Boil opened by crucial incision under ether, which was again taken normally. The pus grew abundant *staphylococcus aureus*. After this there was very little discharge from the wound, which healed rapidly.

Sept. 2. Legs abducted more till adductors just tense. Only tiny granulating area left on buttock.

Sept. 3. 6.45 a.m. Complained of a little pain in left foot; nil found to account for it.

7.30 a.m. Bed made and nurses noticed nothing wrong with the child.

8 a.m. Day Sister passing, noticed a curious blank expression on her face and on examination found her unconscious, pale, sweating freely. The pupils were widely dilated, there was no conjunctival reflex, and her pulse was very weak but not irregular.

A few minutes later, the eyes were rolled upward, and there was a little twitching of the right side of face. She had difficulty in swallowing some brandy which was given, as if, Sister said, she had a spasm of the pharynx.

A quarter of an hour later could swallow, but kept murmuring "No more." Pupils became pinhole. Back was slightly arched and neck stiff. No vomiting or incontinence.

8.40 a.m. Seen by one of the writers. (M.F.B.)

Child drowsy, but responded to request to put out the tongue; then kept on putting it out at intervals. Pupils small, but acted to light. No squint or nystagmus. Abdomen not distended. Skin warm, not flushed. Taken off frame.

9.40 a.m. Seen by second writer (R.G.).

Child still drowsy, but could be roused. Complained of occipital headache; slight rigidity of neck. Pupils equal and active; no squint or nystagmus, no Babinski reflex, all tendon jerks brisk. Urine showed no abnormality. Lumbar puncture advised.

12.30 p.m. Lumbar puncture. Free gush of 40 c.c. of clear fluid. When this was withdrawn, fluid only dripped out during crying. Pathologist's report on fluid: cells under 5 per c. mm.; other characters quite normal.

Widal negative for typhoid and paratyphoid; urine sterile. Fæces: no organisms of typhoid group; no non-lactose-fermenting *B. coli*; no pathogenic streptococci.

Symptoms disappeared almost entirely directly after lumbar puncture, the child talking rationally and taking nourishment.

Sept. 5. All symptoms gone and no abnormality made out except a little twitching of right hand and weakness of its grip, while the tendon jerks of right limbs were a little less brisk than those on left side. Constipation and coated tongue.

Sept. 6. After seeming normal in morning, about 12.30 p.m. began to complain of headache, and temperature rose to 100°. No other change in condition. After this, condition appeared normal, except that temperature remained up to 99° for three days, after which it rose up to over 99° almost every night.

Sept. 25. Frame re-applied, without much abduction. This was not followed by any reaction.

Sept. 28. Frame abducted two holes. Pain and rise of temperature to 100° in evening, so abduction reduced.

Oct. 8. Temperature at 100° at night with nothing to account for it. After this the frame was abducted once without reaction, but on the second occasion this was followed by a rise in the temperature to 99.8°, about which it remained for three days; a further abduction sent it up to 102.6°, while an immediate fall followed diminution of the abduction.

Since that, increase in the abduction has twice been followed by small temperature reactions, and once by none.

Nov. 26. She complained of occipital headache, which was relieved by aspirin and potassium bromide, without development of further symptoms, nor did this follow any interference with the frame.

DISCUSSION.

It seems obvious by a study of the temperature chart of the above case, that whenever the adductors of the hips were put on the stretch, with two exceptions, a mild febrile reaction followed the manipulation, however gently it was carried out. The meningismus, with proved excess of cerebrospinal fluid, appears to have represented the first of these reactions.

If there is a definite connection with the use of an abduction splint, it is curious that those who have had large series of these cases under their care for many years should not have come across and recorded any.

Professor Osgood of Harvard, in a personal letter, states that though he has noted the liability of spastic cases to shock in connection with severe stretching of the muscles round the hip-joint, yet he has never seen meningeal symptoms in such cases.

It may be mentioned that a sporadic case of scarlet fever had developed in the hospital within two weeks before, and another a similar period after, the attack of meningismus recorded here, but this child was not exposed by direct contact with either and never showed the slightest symptom of this disease. Moreover, Dr. Caiger has been kind enough to tell us that in all his experience he has never seen meningeal symptoms form the only manifestation of scarlet fever, unassociated with some at least of the other typical symptoms.

Great care was taken to exclude all possible toxic influences in this case.

1. *General infections.*—The child has had no general infection either before entry to hospital or since.

2. *Local infection.*—A carious tooth was removed a fortnight previously, but there was no apical abscess associated with this.

A boil had been opened five days previously and was only represented by a small clean granulating surface at the date of the attack.

3. *Intestinal tract.*—No disturbance; constipation followed but did not precede the attack. Examination of blood and stools entirely negative.

4. *Urinary tract.*—Examination negative.

5. *Upper respiratory tracts.*—Examination negative.

6. In an open-air hospital in summer, the question of sun-stroke must be considered. On the day before the attack the child was not put outside and there were only 3.8 hours of sun that day. Maximum shade temperature 78.4°.

If toxæmia or infection can be ruled out, is there any other possible explanation?

Professor Osgood has noticed that in certain cases sudden stretching of the adductors produces shock. Recent opinions on this difficult subject appear to agree that one of the causative factors is a profound disturbance of vasomotor control, as a result of noxious afferent impulses. It may be possible that, under slightly different circumstances, the balance in pressure between the blood and the cerebrospinal fluid within the skull is disturbed, and with a too rapid recovery of the systemic vascular tone, the absorption of cerebrospinal fluid cannot restore the balance sufficiently rapidly to prevent symptoms of temporary intracranial pressure arising. This would explain the immediate relief on removing pressure by lumbar puncture and the fact that in less severe cases the symptoms gradually disappear spontaneously even if lumbar puncture is not resorted to.

As this case is of some theoretical and practical interest, it is hoped that neurologists and orthopædic surgeons will combine to throw some light on what appears to be a baffling and somewhat disturbing condition.

CIRCUMCISION AND THE ABREACTION OF FEAR.

By ESTELLE M. COLE, London.

Among war neurotics fear abreactions, either spontaneous or brought about in the course of psychotherapy, were by no means infrequent. The abreaction here dealt with, which occurred in private practice, will illustrate this psychological mechanism and is considered of sufficient interest to be briefly recorded. The patient, a medical man, single, age 31, diagnosed as a case of hysteria, had been undergoing treatment interruptedly for two years. A week previous to the day of the abreaction he complained of loss of interest in his work, inability to work, and great fatigue. These symptoms he thought might be due to certain defects in his metabolism, and he asked leave to consult an investigator in endocrinology, with a view to having his basal metabolism tested, in order that it might be ascertained which glandular extract would correct the deficiency and supply the need for his physical being. To this I assented, since these investigations could not interfere with the analytical work, although I realised that the symptoms of which he complained at that time were due to unconscious mental factors rather than to his metabolic processes.

The investigations took place about thirty-six hours before the abreaction occurred. The patient left the analytical treatment that afternoon (Wednesday) apprehensive at having to undergo these investigations, although, consciously, he displayed interest in them and they were of his own choice. The preparations for them needed retirement to bed at an early hour (7 p.m.), and abstinence from food for 12 hours. The night was an interrupted one, fear kept him awake, he was restless and slept intermittently. No dreams were recollected.

The following morning (Thursday), at 7 o'clock, the physician entered the patient's room bearing the necessary apparatus. The pulse registered 45. The apparatus consisted of a bag, which was placed over the patient's face; he was then told to breathe quietly, and within a minute he felt suffocated and made signs that the bag must be removed. He was reassured and the bag replaced. Breathing was quiet for eight or nine minutes and, the bag being removed, the investigations ended. He was given a meal and told that he could resume his medical duties. There was restlessness and uneasiness, and the remainder of the day was spent in attending to his medical work with great difficulty. He gave way to fits of crying and felt as if some great disaster were about to befall him, and the words "it is all up with me;" "I am done for now," kept on repeating themselves. The throat and tongue felt parched

and dry, and waves of fear were experienced. That night (Thursday) was again restless. He was unhappy and apprehensive of some approaching unknown disaster. No dreams were remembered. The following morning (Friday) there was further difficulty in attending to medical work, and at 3 p.m. he entered the consulting room for his analytical treatment. The facial appearance was haggard and anxious, with dark, deep shadows under the eyes, and his manner was restless. His greeting was serious. He put himself into the supine position and began to describe the investigations, when suddenly he ceased to talk; a violent agitation of the limbs was in progress.

With his hands covering his face, he cried out in great anguish "Oh! Doctor, it is all up with me now," "Even *you* cannot help me." Then, very piteously, "I am done for now, Doctor!" The face and lips were blanched, the tremors were still violent, the whole frame shook, and the forehead looked moist. The voice was one of piteous distress, and he was calling for help. I felt it was time to take his pulse, and under the guise of reassurance, I held his wrist, which felt cold and moist, while the pulse registered 52. My movement gave him confidence. The colour began to return and the hysterical weeping gave place to the long drawn sobs of a small child and gradually ceased. I had returned to my chair behind him. There was silence for a couple of minutes, when he remarked "I feel better now."

This was an abreaction of fear and had lasted two-and-a-half minutes. He then explained in detail that when six years old, he was awakened one morning and given a light breakfast at 7 a.m. "Everyone seemed very kind," he said, particularly his mother and his aunt, neither of whom usually took any notice of him. He grew suspicious of these attentions and thought that something was about to happen. Soon he was wrapped in a blanket and carried downstairs attended by his old nurse, mother and aunt. Suddenly he was placed upon a table and left alone. Two men (surgeon and anæsthetist), dressed in black, appeared on the scene. One produced a bag with which he covered the patient's face. There was a short struggle and the words "it is all up with me," "I am done for now," passed through his mind, and the patient knew no more. After some hours he awoke, to find himself in bed with a pain in the genitalia. He had been circumcised for bed wetting.

The hour of treatment was now at an end and he left the room feeling that his fear had subsided. He had lost his anxious look, and smiled his thanks for the help he had received.

It is a simple matter to correlate this abreaction with the incidents that provided the stimulus, i.e., the endocrine investigations.

Editorial.

PROGNOSIS IN THE PSYCHOSES.

AT the present time scarcely any diseases are the subject of more investigation than mental disorders, and every endeavour is now being made to utilize any methods of treatment, both biological and psychological, which may lead to their amelioration or cure. In spite of this widespread activity, however, and in spite of the often expressed wish on the part of the psychiatrist that the psychoses should be regarded as naturally and hopefully by the general public and the medical practitioner as other types of illness, an attitude of pessimism in regard to insanity is the rule rather than the exception. Many factors no doubt contribute to the development of such an attitude, one of which is the widespread belief that the psychoses are incurable. There are, indeed, distinguished psychiatrists who would hesitate to use the word "Cure" otherwise than in inverted commas. It does not follow that a pessimistic point of view towards any particular problem is necessarily unjustified, and there are certainly features in mental disorders which tend to foster a gloomy view as to their outcome. Thus, segregation is necessary for a large number of psychotics, and the unrecovered patients are therefore more prominently displayed to the gaze of the public, as it were, than is the case with chronic patients suffering from other forms of illness. In view, then, of the popular impression that mental cases go from bad to worse, and that when they "recover" they break down again, it is most desirable that attempts should be made to discover what really does happen to patients when they leave mental hospitals. It is probably true to say that the prognosis for the general run of mental cases has never been established, and for this reason attention may be drawn to a concise and timely contribution to this problem by Dr. Earl D. Bond[1], who writes an article on the underestimation of good results in mental diseases. Beginning in 1914, the careers of 1,054 consecutive patients admitted to the department for mental and nervous diseases of the Pennsylvania Hospital were studied

for a period of from five to ten years. The majority of the patients were suffering from the more severe types of mental disease, only a small proportion being classified as psychoneurotics; of these consecutive admissions, thirty were lost; of the 1,024 patients that could be followed over five years, 26.7 per cent. recovered and stayed well; 14.5 per cent. improved; 32.3 per cent. died; and 25.5 per cent. did not improve. The writer comments on these groups with only one point in mind, namely, the prevailing tendency to underestimate good results. In the first place, among those who recovered are some patients who reached a higher level of health than they had been able to reach before the illness. Thus, a woman who had been doing mediocre work as a shop sales clerk before her illness, was able to take up college work, graduated from a school of dietetics, and has for some years been chief dietetician in a large general hospital, following her recovery. Such cases are of considerable psychiatric interest and are by no means infrequent. The psychosis in these patients appears to have a cathartic effect—it 'clears the air,' as it were, and affords a release of pent-up emotional tension. In the second place, a number of paranoid conditions in which it is customary to assign an unfavourable prognosis are noted as having cleared up, as well as psychoses exhibiting severe catatonic symptoms.

As regards the "improved" cases attention is called to the fact that some patients are placed in this category because their return towards health was only partial, and others because they relapsed. Any cases which relapsed during the period under review were excluded from the "recoveries" even though the patients became normal again. With a few exceptions this group is composed of hard workers, and the improvement reached the state of self-support. The death-rate in this series appears large on the surface, but following any group of one thousand persons, many of them in the later decades, even in a space of five or ten years would bring to light a considerable mortality. As the period of follow-up work runs through 1918, there were a number of deaths from influenza. Many of the causes of death were not related to the psychoses in any way. On the other hand, all the patients with senile and arteriosclerotic psychoses died during the follow-up period.

Unfortunately it is not possible to compare these figures with similar ones in relation to physical diseases, for, as Dr. Bond observes, we have almost no information as to what

happens to patients who come consecutively to the doors of any surgical, medical or special hospital, or to the consulting rooms of general practitioners or specialists. There are plenty of statistics about patients with this or that disease, but none about the general run of patients. Probably the surgeon or physician would be satisfied with a return to full function in 25 per cent. of his consecutive cases, judgment being made from five to ten years after the beginning of treatment. Everything in psychiatry to-day points to the prospect of increasing the recovery percentage by getting at mental diseases early. If by getting at them late, consecutive cases may be expected to show full return to health in 25 per cent., and amelioration in 15 per cent. more, the general practitioner first, and the psychiatrist later, are justified in taking as hopeful an attitude as is taken for surgical problems.

These facts are deserving of emphasis if only for the sake of recovered psychotics who are, in many instances, only too conscious of the fact that they are at a considerable social disadvantage on account of their illness. Those who have an intimate knowledge of these patients cannot fail to be impressed by their extreme sensitiveness in regard to the attitude of the outside world towards them—a reaction which sometimes causes them to dread the time when they will have to leave the mental hospital. It is thus desirable that there should be a more general recognition of the fact that the psychotic may both become and keep perfectly well. Furthermore, though we have no knowledge of any specific remedies which will cure a psychosis, there are features in this form of illness which suggest that the discovery of such remedies is not beyond the bounds of possibility. Spontaneous cures occur in the psychoses which have no parallel in purely physical diseases of the chronic type. Such cures appear almost miraculous and astonish all concerned in the care of the patients in whom it is exhibited. Thus every psychiatrist could furnish instances of patients who have (apparently) been in a state of hopeless deterioration for many years, and who gradually come to themselves and make a complete recovery. In other cases a physical illness may be responsible for the amelioration or cure of a psychosis—a reaction suggesting thereapeutic measures which have not been altogether unsuccessful. As an instance the malarial treatment of general paralysis may be cited.

We do not know much about the biological disturbances which are responsible for prolonged mental illness, nor do we

know what happens within the organism to produce a return to normality. The fact, however, that patients with these serious mental illnesses are capable of recovery is one of profound psychiatric significance. Since such patients can recover, that is to say there is no irreparable structural change which renders a return to the normal an impossibility, it is certainly permissible to indulge in the hope that further investigation may not only enable us to understand why they do so, but that it may also suggest directions in which therapeutics may be applied with a view to hastening or bringing about the natural processes of recovery. There would seem to be no valid reason why the treatment of the psychoses should not one day be as successful as that of any other diseases. Perhaps more so, indeed, since we seldom meet with such "miracles of healing," as those referred to above as occurring in the psychoses, in any other morbid conditions. Thus we can scarcely expect a patient suffering from (say) chronic osteoarthritis so to improve as to exhibit a full return to function. In the first place, then, it would appear that the prognosis in the general run of mental disorders is by no means so unfavourable as some would believe, and, in the second, it would seem to be reasonable to nourish the hope that, with increasing knowledge of the causation and treatment of these disorders, it will be still more favourable than is at present the case.

* *Journ. Amer. Med. Assoc.*, Aug 15, 1925.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[136] **Studies on the central visual system. 1. The anatomic projection of the retinal quadrants on the striate cortex of the rabbit.**—T. J. Putnam and I. K. Putnam. *Arch. of Neurol. and Psychiat.*, 1926, xvi, 1.

In the rabbit there is a fixed and definite anatomical projection of the dorsal nucleus of the corpus geniculatum externum on the cortex, and lesions of the striate cortex only a few millimetres in diameter cause circumscribed and sharply localised areas of atrophy in the external geniculate. Contiguous lesions of the cortex produce contiguous lesions in the corpus geniculatum, and the size of the one is proportional to the size of the other, in the areas devoted to monocular vision. On the other hand, lesions of the striate cortex in the adult rabbit cause no demonstrable lesions of the ventral nucleus, the anterior colliculus or the optic tract. When the results of these experiments are combined with the already known projection of the retina on the corpus geniculatum, the nasal quadrants are found to be projected on the postero-inferior portion of the striate cortex, and the temporal quadrants on the antero-superior portion. The field for binocular vision has a relatively large representation anterior to that of the temporal quadrants. The superior retinal quadrants are represented superiorly in the cortex, the inferior quadrants inferiorly; so that a second rotation of the field must take place in the geniculocortical neurone.

R. M. S.

[136a] **Studies on the central visual system. 2. A comparative study of the form of the geniculostriate visual system of mammals.**—T. J. Putnam. *Arch. of Neurol. and Psychiat.*, 1926, xvi, 285.

The primitive mammalian type of corpus geniculatum externum, with a dorsal and ventral nucleus, without lamination, lying vertically on the surface of the brainstem, is seen in marsupials and lower mammals. Only the dorsal nucleus has a projection on the cortex and an indication of this is seen in the brain of the didelph. Of the animals studied, the cetacea, carnivora and primates showed a variation from the primitive mammalian type of corpus geniculatum. This consists of the almost complete disappearance of the ventral nucleus; the appearance of one or two rows of large cells along the periphery of the persisting dorsal nucleus; certain deformations of the primitive

form, perhaps resulting from an increase in the size of the area of representation of binocular and macular vision; and a rotation lateralward, so that the original external surface lies ventrally. In all the animals studied, the fasciculus longitudinalis inferior appeared to be the only system connecting the external geniculate body with the cortex, and in some animals (*phoca*, *phocæna*) the entire fasciculus can be unmistakably traced to it.

With the other reasons for believing that the inferior longitudinal fasciculus is the geniculostriate radiation should be considered the fact that its fibres resemble those of the optic tract, and also other important radiations. Such coarse fibres are not seen in association systems. In the ape, fibres are given off only from the superior and inferior edges of the longitudinal fasciculus and simultaneously to the lateral and mesial cortex. This speaks in favour of the "vertical division" of the optic radiation. The existence of callosal fibres uniting the corpus geniculatum with the opposite cortex has been postulated on hypothetical grounds, but the author could find no evidence of decussating cortical fibres in the present study.

R. M. S.

[137] **The radix spinalis trigemini and the principle of usurpation.**—Walter Freeman. *Arch. of Neurol. and Psychiat.*, 1926, xv, 607.

One of the interesting features of the trigeminal nerve is its long caudal extension in the radix spinalis. The axons of the cells in the gasserian ganglion enter the pons and divide, one branch going to the main sensory nucleus, the other turning caudad and extending even as far as the first or second cervical segment of the spinal cord. The behaviour of these fibres is different from that of the homologous fibres in Lissauer's tract in the spinal cord. In the spinal cord these fibres run for a distance of one or at most two segments before entering the gray matter of the posterior horn, whereas in the case of the spinal trigeminal tract they run for a much greater distance before terminating in the substantia gelatinosa of the bulb. The explanation for this is to be sought in the phylogenetic development of the trigeminal nerve.

The fifth nerve is not the only one which contributes fibres to the radix spinalis trigemini. The seventh, ninth, and tenth cranial nerves also contribute to this root in the lower fishes, so that the similarity of the root to Lissauer's tract is quite marked. During the development of the head these lower branchial nerves have gradually lost their cutaneous fibres, and the trigeminal nerve has increased in importance until it has covered all of the face and a large part of the head. Why there has been this progressive concentration of function in a single nerve is difficult to explain, except that concentration and centralisation seem to be in keeping with the economy of the organism. Clinical investigation has also shown that the substantia gelatinosa of the brainstem is segmented in a primitive fashion, focussing on the mouth, just as the lower sacral segments of the cord focus about the anus. With

these two facts in mind, (1) that the trigeminal nerve has usurped the fields formerly supplied by several other nerves, and (2) that there is definite segmental representation in the spinal root of this nerve, we may arrive at an explanation of the long caudal projection of the afferent fibres. Pain, pressure, heat and cold are primitive types of sensibility accompanied by a high degree of feeling-tone. They might be called palæo-æsthetic sensibility. The receiving centres for these sensations are arranged in primitive fashion, segmentally in the substantia gelatinosa rolandi in the medulla oblongata just as in the spinal cord. These centres, laid down early in the course of phylogenesis, do not change their position. Therefore, when a single nerve usurps the function of several other nerves, the fibres supplying the invaded area must travel a greater distance within the nervous axis in order to reach the cells of origin of the secondary pathway. In the case of the trigeminal nerve this probably explains the long caudal projection of the axons of cells in the gasserian ganglion.

R. M. S.

[138] **The development of the third nerve nucleus and its bearing on the Argyll Robertson pupil.**—Leslie Paton and Ida C. Mann. *Trans. Ophthalm. Soc.*, 1925, xlv, 610.

Summarising the results of their researches, the authors conclude as follows:—

1. The occurrence of the Argyll Robertson pupil depends on a break in the reflex arc in that part consisting of the intercalated colliculonuclear neurone and the nucleus of origin of the efferent irido-constrictor fibres.

2. The origin and path of the colliculo-nuclear neurone concerned have not yet been defined, but hypothetically it has its cell in the fifth stratum of the colliculus, and its tract either in the inner layer of the arcuate fibres or in the fibræ radiales.

3. The fibræ radiales give the most direct connexion with the Edinger-Westphal nucleus.

4. Evidence (developmental) is adduced to show the relationship of the Edinger-Westphal nucleus with the rest of the third nucleus, and to show that its differentiation from the third nucleus synchronizes with the development and functioning of the sphincter pupillæ musculature, both phylogenetically and ontogenetically.

S. A. K. W.

[139] **Review on the tonus of skeletal muscle.**—Stanley Cobb. *Physiol. Reviews*, 1925, v, 518.

According to Sherrington, reflex tonus is reflex posture, "a reflex which differs from the reflexes more commonly studied mainly in this, that the latter execute movements while this maintains posture." Thus viewed, tonus is that state

of constant muscular tension which underlies and makes possible all orderly motion.

1. *The question of sympathetic innervation.* Though the subject is far from settled, the author states we are justified in concluding, in the words of Langley, that " there is a balance of histological evidence that sympathetic nerve-fibres form hypolemmal endings on some striated muscle fibres."

2. *Innervation of red and white muscle fibres.* It appears that in mammals red and white muscles differ somewhat both in anatomy and physiology, but the distribution is variable and white fibres may perhaps turn red. The type of innervation gives no clear and consistent anatomical basis for distinguishing between red and white fibres. There appears to be, however, a clearer distinction in reptiles.

3. *Contractility of sarcoplasm.* The author cannot find that there has ever been any proof that the sarcoplasm in a muscle fibre is in fact contractile. It is purely a hypothesis.

4. *Sympathetic influence on muscle tone.* After a long examination of the data advanced by Hunter, Langelaan, and many more, the author summarizes as follows : " until it can be proved (*a*) that mammalian muscle contains two distinct forms of fibre, the slender and the coarse, each with a special innervation, and until (*b*) there is satisfactory proof of the existence of two kinds of muscle tone, plastic and contractile—the theory of Hunter cannot be accepted. Additional evidence is also desirable to explain the ' interval ' after operation. Nor will the evidence ever be satisfactory until some positive proof is obtained through stimulation experiments."

5. *Creatin metabolism.* No satisfactory evidence has been produced that creatin metabolism is in any special way associated with tonic muscular contraction.

6. *The tonus of skeletal muscle as a proprioceptive reflex.* Static and kinetic movements can be explained on the basis of one neuromuscular mechanism. ' Tonic ' and ' phasic ' reflexes, though unlike, are separated by no fundamental difference in physiological mechanism. Tonus is a graded series of proprioceptive reflexes, continuously and unconsciously playing its part in every motor act. By its remarkable specificity it moulds individual muscles ; by its universality it controls postures.

S. A. K. W.

NEUROPATHOLOGY.

[140] **A contribution to the pathology of paralysis agitans.**—C. M. Byrnes. *Arch. of Neurol. and Psychiat.*, 1926, xv, 407.

This paper is devoted to a study of the muscular tissue and its proprioceptive system of nerve terminals in paralysis agitans, and is well illustrated by numerous drawings and microphotographs, some of which are in colour. Byrnes considers that there is much in the clinical course of paralysis agitans

to suggest that in some instances, at least, it may be dependent on a morbid process situated in the peripheral mechanism concerned in the production and maintenance of muscle tone, and accordingly he made an examination of the muscular tissue obtained by biopsy from fourteen cases of genuine paralysis agitans. No evidence could be obtained that the disease is of myopathic origin, but a constant lesion in the neuromuscular bundle was discovered, characterised by œdema, early degeneration of the intrafusal nerve, and final disintegration of the entire spindle. The intramuscular nerve trunks were normal, and the surrounding musculature was, as a rule, unaltered. Although studied in a variety of other diseases, no lesion of the neuromuscular bundle similar to that observed in paralysis agitans was discovered, and eight muscle spindles from a case of postencephalitic Parkinsonian syndrome of five years duration appeared perfectly normal. It is therefore legitimate to assume that the lesion of the spindle in cases of paralysis agitans is primary, and not secondary to the tremor or the rigidity. It appears to be a toxic parenchymatous degeneration of the intrafusal nerve terminals, and it is conceivable that one or more toxic substances of metabolic or glandular origin might exhibit a selective affinity for the neuromuscular bundle. In view of the fact that paralysis agitans is not accompanied by any appreciable abnormality in the perception of muscle sense, Byrnes concludes that the spindle does not subserve this function. It does, however, play an essential part in the mechanism of muscle tone, and it is the author's belief that the spindle is the afferent inhibitor of the striothalamic tonectic centres. Hypertonia of the Parkinsonian type might originate, therefore, from either central or peripheral disease of this system of fibres; and it is suggested that the central lesion finds its chief exponent in the postencephalitic syndrome, while genuine paralysis agitans in its typical form is characteristic of the peripheral lesion. This assumption does not preclude, however, the possibility of a combination of the two lesions in certain types of Parkinson's disease in which the symptoms are widespread and fully developed in the early course of the disorder.

R. M. S.

[141] **The spinal fluid sugar in cases of epidemic encephalitis and other nervous disease** (Recherches sur la glycorachie dans les cas d'encéphalite épidémique et d'autres affections du système nerveux central).—C. J. Munch-Petersen and Knud Winther. *Acta Psychiat. et Neurolog.*, 1926, i, 188.

Using the method of Hagedom-Norman-Jensen, the authors have investigated the sugar content of the cerebrospinal fluid in various neurological disorders. They found a variable degree of sugar increase in the acute (rarely the chronic) stage of encephalitis; sometimes there was a corresponding increase in the blood sugar level. This hyperglycorachis is not diagnostic of encephalitis, being found in many other pathological states (diabetes, malaria,

certain fevers and intoxications, cerebral and spinal tumours, epilepsy, paralysis agitans, cerebral softening). According to the literature an increased sugar value has also been noted in acute poliomyelitis, dementia præcox, general paralysis and syphilitic meningitis.

The authors believe, however, that an estimation of the spinal fluid sugar may be of value in differentiating epilepsy from hysteria, and meningitis from meningismus. In meningitis, examination of the spinal fluid sugar from day to day may be a prognostic aid, a return from a low figure to normal being a good omen.

M. C.

[142] **Vegetative disturbances in postencephalitic syndromes and their relationship to diencephalic and mesencephalic lesions: changes in basal metabolism.**—S. NACCARATI. *Jour. Nerv. Ment. Dis.*, 1926, lxiii, 219.

THE basal metabolic rate was determined in 47 cases of postencephalitic syndrome. Of these 53 per cent. gave a plus reaction, seven per cent. a minus reaction, while 40 per cent. were within normal limits. At the same time other vegetative disorders such as obesity, polyuria, etc., were noticed. The explanation given is that there is a complex system of vegetative centres controlling both sympathetic and parasympathetic activities in the midbrain and basal ganglia, and that varying lesions destroying or obstructing, now one path or centre and now another, account for the variations in the symptoms.

R. G. G.

SENSORIMOTOR NEUROLOGY.

[143] **Tumours of the frontal lobe** (Les tumeurs du lobe frontal (abcès exceptés): contribution à l'étude clinique de ce lobe.)—P. ESCUDER NUÑEZ. *L'Encéphale*, 1926, xxi, 37.

THE author reviews the literature dealing with the symptomatology of frontal lobe tumours and describes two fresh cases, one a case of glioma of the right frontal lobe, with a smaller tumour of the left temporal pole: the other a case of hydatid cysts in the left frontal and the parietal lobes. Of the many symptoms considered to be to some extent diagnostic of tumours of the lobe the author lays special stress on deviation of the head and eyes to the side opposite to that of the tumour, or paralysis of conjugate deviation of the eyes to this side. Of the psychic changes the most striking is "hyperexcitation of the imagination," which over-rides other faculties such as memory and attention and often leads to an irrepressible optimism. One proof of this hyperexcitation is an exaggerated response to pinprick, only present when the patient watches the pin. It often leads to alterations of conduct, such as

dirty habits and rudeness of speech ; urination in bed, on the floor or in public is common. (The author applies the Spanish word "atolondramiento" (thoughtlessness) to this condition). This excitement of the imaginative faculties, or "psychical epileptoid state," is a symptom of the earlier stages, and later gives place to a condition of psychical inhibition, somnolence, despondency, dullness, stupor ; finally a kind of coma vigil sets in to which Milian gave the name of frontal coma.

J. G. GREENFIELD.

[144] **Cerebral tumour [glioblastoma] with early psychasthenic syndrome** (Tumeur cérébrale (glioblastome) avec syndrome psychasthénique initial).—L. MARCHAND and P. SCHIFF. *L'Encéphale*, 1926, xxi, 121.

THE case described is that of the capable manager of a factory, who developed a tumour in both frontal lobes and the inferior part of the genu of the corpus callosum. The first symptoms which he presented were progressive slowing down of intellectual activity, melancholy, anxiety, scrupulosity, obsessions of doubt, a mania for order, and intellectual fatigability. He also had occasional conscious incontinence of urine, and sometimes attacks of trembling. Nocturnal attacks of headache, sometimes assocated with vomiting, came on later. Apart from a doubtful congestion of the left optic disc he presented no objective signs of disease. The diagnosis of cerebral tumour was, however, confirmed by the results of lumbar puncture, which showed a greatly increased cerebrospinal fluid pressure. A right frontal craniotomy was performed without a tumour being discovered. He developed a cerebral hernia and died five months after the operation.

The tumour was found to involve the greater part of the anterior half of the left cerebral hemisphere from the cortex to the anterior third of the thalamus. It passed across the middle line by the inferior part of the genu of the corpus callosum and surrounded the anterior part of the right lateral ventricle.

Histologically the tumour was a glioma of a mixed type presenting in some areas huge fibrous astrocytes and in others masses of undifferentiated rounded or elongated cells.

The case is of interest as a contribution to the clinical syndrome of tumours of the frontal lobes and corpus callosum.

J. G. GREENFIELD.

[145] **The anatomy and clinical aspects of pure word-deafness** (Beitrag zur Anatomie und Klinik der reinen Worttaubheit).—P. SCHUSTER and H. TATERKA. *Zeits. f. d. g. Neurol. u. Psychiat.* 1926, cv, 494.

THE patient was a man of 67 who developed a right hemiparesis and some eighteen months later had an attack followed by loss of appreciation of words, sounds and melodies : these symptoms remained unchanged to the date of

his death, after two more years. The clinical condition, more exactly, was as follows: intelligence conserved, with full insight into his own condition; hearing normal on both sides; understanding for speech practically lost, with minor exceptions: repetition of words impossible, or nearly so; spontaneous speech relatively quite good, with occasional paraphasia; reading aloud, and understanding of written speech, normal; spontaneous writing quite intact (left, mirror writing); inability to write to dictation; appreciation of melody greatly interrupted; mild auditory agnosia otherwise.

The pathological examination, conducted in full detail, gave a paraventricular softening in the left hemisphere, extending along the outer aspect of the ventricle from the posterior end of the capsule backwards; softening in the claustrum and in the gyri of Heschl, involving the white matter underneath the posterior third of the first and second left temporal gyri; the cortex of the latter was intact.

This case is of very considerable scientific interest, in view of the pureness of the clinical word-deafness and the smallness of the temporal lesions, to which with ample justification the authors attribute the speech defect.

S. A. K. W.

[146] **Further observations on involuntary grasping and analogous disorders of movement** (Weitere Beobachtungen über Zwangsgreifen und Nachgreifen und deren Beziehungen zu ähnlichen Bewegungsstörungen).—Paul Schuster and Hermann Pineas. *Deut. Zeits. f. Nervenheilk.*, 1926, xci, 16.

The authors have here recorded a number of observations bearing on the difficult symptom of so-called tonic innervation, or inability to relax a given voluntary innervation once it is initiated. They have found this phenomenon in various cases exhibiting, in addition, symptoms of an extrapyramidal kind, with pathological changes in thalamus, corpus striatum, or frontal cortex, or in both cortex and basal ganglia. They are of the opinion that factors of an unconscious kind, as well probably as a psychical component, should be taken into etiological consideration. They cannot say what cerebral, non-pyramidal, mechanisms must be involved for the symptom to occur, and are disinclined to associate it with any specific lesion; they think, rather, that it is caused by disorder of a " constellation " of movement, that is, of the various aggregates of normal movement and the corresponding conduction paths.

The authors find themselves in agreement with much of what Wilson and Walshe have previously published on this question (they quote the paper as having been printed in 1924, whereas 1914 was the year of publication), but they have widened the basis of the symptom on the clinical side and their own conclusions, they candidly admit, are somewhat indefinite. The subject is one requiring much more study.

S. A. K. W.

[147] **Bulbar syndrome predominantly interolivary; progressive vascular changes with hypoplasia and neuromata of the medulla and spinal cord** (Syndrome bulbaire à prédominance interolivaire; altérations vasculaires progressives, hypoplasie et névromes bulbo-médullaires). —G. MARINESCO and S. DRAGANESCO. *L'Encéphale*, 1926, xxi, 89.

THE case described was clinically one of quadriplegia. The patient at the age of 50 began to feel weakness in the left leg, and this progressed and spread gradually so that three years later he could scarcely walk. By this time weakness and paræsthesia of the right arm had appeared. Two years later the right leg and left arm were also affected in a similar manner and he became bed-ridden. He had also some difficulty in swallowing. On examination at this time some weakness of the left side of the face was noted. The pupils were myotic but reacted to light. He was deaf in the left ear with tinnitus. The arms were very weak and rather hypotonic, the lower limbs spastic with involuntary reflex movements. The spinal fluid was normal and the Wassermann reaction in the blood negative.

On examination three years later (1923) there was definite atrophy of the left side of the tongue with weakness of the left side of the soft palate and at times aphonia. Wasting of the small muscles of the right hand was also present and the diagnosis of atypical amyotrophic lateral sclerosis was made.

On post-mortem examination the arteries of the base of the brain were very atheromatous. Minute softenings were found in the lenticular nuclei and in the pons. The medulla was smaller than normal and the prominence normally formed by the pyramids was flattened down. The cord was also shrunken. The chief histological changes in the cervical cord were: (1) minute neuromata, resembling amputation neuromata, in the leptomeninges on the posterior surface of the spinal cord, chiefly round the blood vessels; (2) areas of areolar degeneration of the posterior part of the dorsal columns and in the region of the pyramidal tracts resembling that seen in subacute combined degeneration; (3) intense atheroma of the blood vessels and considerable thickening of the meninges. Similar changes were found in the lower part of the medulla, but here the neuromata were found also along the dorso-median fissure and passing into the columns of Goll. The pyramids and the fibres in the hilum of the olives, as well as those of the mesial fillet, were severely degenerated and the arcuate nuclei were destroyed. There was also areolar degeneration in the region of the restiform bodies and the descending root of the trigeminal. These changes became less on passing upwards, so that in the uppermost part of the medulla they were comparatively slight. Elsewhere in the nervous system no changes other than the small softenings already noted were discovered.

In reviewing the case the authors consider that the majority of the lesions might be explained by the intense atheroma, which had led to gradual reduction

of the blood supply and to complete blockage of one or two vessels. But they consider that the general atrophy of the medulla and cord and the neuromata suggest a congenital basis for the condition. They are, however, alive to the possibility that the atrophy might be due to ischæmia and the neuromata to regeneration of damaged posterior root fibres.

J. G. GREENFIELD.

[148] **A rare form of contracture of the limbs in a case of postencephalitic Parkinsonism** (Intorno ad una rara forma di contrattura degli arti osservata in un malato affetto da parkinsonismo post-encefalitico). —L. MAGNI. *Riv. di pat. nerv. e ment.*, 1926, xxxi, 46.

A REVIEW of a case of contracture of flexion of the lower limbs with coincident equinus, interesting because observed in a case of postencephalitic Parkinsonism and because it became permanent and irreducible in the short period of three months. This was supposed to be due to a mesencephalic lesion involving both the tract from the corpus striatum to the locus niger and the cerebral peduncle, with coincident degenerative phenomena in the spinal motor tracts. Such contractures may be due not only to cortical or medullary lesions but also to mesencephalic lesions.

R. G. G.

[149] **Tonic eye fits ("oculogyr crises") in chronic epidemic encephalitis.**—AUGUST WIMMER. *Acta Psychiat. et Neurolog.*, 1926, i, 173.

OCULOMOTOR crises constitute one of the less common phenomena appearing after epidemic encephalitis, usually in association with the Parkinsonian syndrome. The attacks consist in a spasmodic deviation of the eyeballs, most often vertically, occasionally downwards. The fits may arise at any time, sometimes as the result of fatigue, at other times during voluntary deviation of the eyes. The influence of emotion, or exposure to strong lights, may at times be noted.

During the attack the patient is quite unable voluntarily to alter the direction of the eyes, even though the fixed attitude may be accompanied by the utmost distress or pain. In duration, the crises vary from a few seconds or hours to the greater part of a day. Sometimes the fits are accompanied by spasms in other muscles, the oculomotor crises in such cases constituting a part merely of more universal tonic fits. In discussing the morbid physiology, Wimmer emphasizes the frequent coexistence of Parkinsonism. He quotes Statz's conception of a topographical representation in the corpus striatum of individual somatic segments including the eye muscles. Wimmer is inclined to implicate the neostriatum as the site of the lesions.

M. C.

[150] **The problems of eye movements in the vertical direction** (Probleme der vertikalen Blickbewegungen).—Otto Marburg. *Zeits. f. Augenheilk.*, 1926, lviii, 253.

The precise localisation of the physiological centres controlling upward and downward movements of the eyeballs is insufficiently known. Tumours in the region of the anterior colliculi unquestionably are calculated to produce disorder of vertical movement, but they are not satisfactory from the viewpoint of localisation on a minute scale. In cases of paralysis of vertical deviation the patient can usually 'roll' his eyes, hence nuclear lesions of superior and inferior recti and of trochlearis are scarcely possible. 'Supranuclear' systems capable of effecting vertical movements belong either to the pyramidal or to Deiters' system. Though we can, of course, move the eyes voluntarily upwards and downwards, cortical and subcortical lesions of the pyramidal paths never appear to produce vertical paralysis. In tracing pyramidal degenerations fibres are practically never found which appear to pass to the oculomotor nuclei at the level of the anterior colliculi: they are seen rather at the level of the most posterior part of the peduncle and anterior part of the pons. The author has shown that vertical nystagmus can develop when the anterior section of the path from Deiters' nucleus to the oculomotor nucleus is involved, and he concludes that under appropriate circumstances paralysis of vertical movement (voluntary or involuntary) will follow lesions situated in an area between Deiters' nucleus posteriorly and the oculomotor nuclei anteriorly.

S. A. K. W.

[151] **The oculo-cardiac reflex; its clinical significance.**—R. D. Gillespie. *Jour. of Ment. Sci.*, 1926, lxxii, 321.

One or two estimates of the reflex are of little significance, and may actually be misleading. It is often very variable in the same individual: and it tends to vary in an irregular manner throughout the day, and from day to day. Values up to three (or more), even if obtained from a considerable series of readings, are often of no significance. For eliciting the reflex, prolonged pressure is more reliable than brief pressure. The reflex passes off in the majority of cases within 20 seconds of the release of pressure. Its slowing down is roughly proportional as a rule to the amount of pressure exerted. No relation was observed in the series of cases between the magnitude of the reflex and the prevailing mood, or between the variability of the reflex and the type or variability of the mood.

C. S. R.

[152] **New studies on tabes** (Sur le tabes: nouvelles études pathogéniques, anatomo-pathologiques et cliniques).—Gonzalo R. Lafora. *L'Encéphale*, 1926, xxi, 161.

In spite of its title this paper does not introduce any new work on tabes, but gives a useful review of the various theories of the pathogenesis of the disease,

The author accepts Richter's views as affording the best explanation, not only of the anatomopathology, but also of the clinical pathology of the disease. According to Richter the disease in a pure form is confined to the nerve roots, and meningitis, when present, is a mere chance concomitant. Consequently the changes in the cerebrospinal fluid which are considered characteristic of tabes (pleocytosis, globulin excess, Lange curve, and Wassermann reaction) are no indication of the activity of the disease, as they merely reflect the degree of meningitis present. Any or all may be absent even when the disease is progressing rapidly. He pleads for the recognition and treatment of tabes in its pre-ataxic stage, even when the spinal fluid is normal, as thorough and prolonged treatment at this stage offers the best hope of cure. If left untreated the disease may at any moment produce symptoms which are more disabling to the patient and more resistant to treatment than the lightning and girdle pains of the earlier stages. The treatment he advises is an initial thorough course of mercury and bismuth in moderate doses. Arsenic should not be used at first as it is apt to increase lightning pains, but may be employed later as a variant to the other remedies. Mercurialized autogenous serum given intrathecally often produces dramatic improvement when intravenous and intramuscular injections appear to have failed, but this method should not be employed until the patient has undergone a course of treatment by the usual routes. The beneficial action of bismuth cannot be explained by its passage into the cerebrospinal fluid, as the most recent experiments in France and Spain have failed to demonstrate any such passage either in animals or in man.

J. G. GREENFIELD.

[153] **The abortive type of Friedreich's disease.**—C. R. ROMBOLD and H. A. RILEY. *Arch. of Neurol. and Psychiat.*, 1926, xvi, 301.

ROMBOLD and Riley record three generations of a family in all of which could be found individuals who at varying times of life manifested a group of symptoms and physical signs which constituted a *forme fruste* of the entire syndrome of 'hereditary ataxia.' This group of symptoms is limited to the lower extremities and constitutes what may be termed an abortive type of circumferential spinal sclerosis or Friedreich's ataxia. The disease begins with some disturbance in gait and the development of the foot deformity, a pes cavus. The process advances sufficiently to destroy the reflex arc controlling the deep tendon reflexes of the knee and ankle, probably through the degeneration of the collaterals which sweep forward from the entrance of the dorsal roots into the spinal cord to end about the ventral horn cells or intercalated cells, thus completing the reflex arc. The process then advances and involves the reflex arc controlling the reaction of the toes to stimulation applied to the plantar surface of the foot, so that the normal plantar flexion is lost and a clear cut Babinski sign, or a fanning reaction, results. The degeneration terminates apparently at this point and the other characteristics of the disease, the ataxia, the scoliosis, the nystagmus and the dysarthria, fail to develop. R. M. S.

[154] **Vagotonia and hypoglycæmic reactions.**—J. M. Nielsen. *Jour. Nerv. Ment. Dis.*, 1926, lxiii, 456.

Vagotonia, beside occurring in psychopathic states and in neurotics, is frequently the end result of a strenous life. It represents a fatigue of the sympathetic nervous system and the endocrines. Hypoglycæmic reactions are very common symptoms of vagotonia and are frequently the incapacitating factor. These are usually weakness, hunger, sweating, insomnia and tremor. Vagotonia may alternate with sympathicotonia in the same patient. Renal glycuresis occurs in about half the cases of this series but is not satisfactorily explained.

Treatment takes considerable time. Of all therapeutic measures, rest is more important than any other. Drug treatment is only palliative. In severe cases the patients manifest an inversion of the law of hypertrophy; they weaken by exercise. The basal metabolism is probably at first high, then approaches zero, finally becoming very low.

R. G. G.

[155] **Migraine brought on by hyperpnœa** (Hyperventilationsmigräne).—Muck. *Münch. med. Woch.*, 1926, lxxvi, 982.

The effect of hyperpnœa was examined on 17 subjects of migraine and on 12 controls. In the controls, beyond paræsthesiæ in the extremities and slight dizziness no abnormal symptoms were produced. In 12 of the migrainous patients more or less typical attacks of migraine were produced within two minutes; in the remaining five the symptoms were similar to those in the controls. The 12 patients in whom attacks were brought on all gave, immediately before the experiment, positive reaction to Muck's adrenalin test, which the author interprets as indicating a tendency to spasm of the vessels, while all the five who failed to respond gave a negative reaction to this test.

J. P. Martin.

[156] **The variability of the blood-pressure in some morbid conditions.**—R. D. Gillespie. *Jour. of Ment. Sci.*, 1926, lxxii, 325.

Three groups of subjects were observed: (*a*) a group showing obvious depressive affect (23 subjects); (*b*) a group not specially characterized by a prevailing affect, and belonging to the schizophrenic type of reaction (15 subjects); (*c*) a group of subjects with organic nervous disease (9 subjects.) There was a considerable variation in the blood-pressure, both systolic and diastolic, in all the groups of subjects observed. This variability was evident in the course of a single day, and also from day to day. The diastolic pressure may, and not infrequently does, vary proportionately as much as the systolic. The variability is greater in restless subjects, under the conditions of the experiment. The variability is greater on the whole in affective disorders and in organic nervous disorders than in schizophrenic cases. The blood-pressure is not necessarily more labile in subjects showing lability of affect than in

subjects in whom the depth of affect does not appreciably vary. It does not follow a uniform diurnal course. When average pressures were considered, it was exceptional for a subject with a prevailing depressive affect to have a systolic blood-pressure persistently above normal: whereas the schizophrenic group showed persistently high and persistently low pressures in about an equal proportion of cases.

C. S. R.

PROGNOSIS AND TREATMENT.

[157] **The arrest of luetic optic atrophy by cisternal injection of mercury.**—Norman Viner and S. O. McMurtry. *Can. Med. Assoc. Jour.*, 1925, xv, 910.

The usual technique for cistern puncture was followed; as soon as the cerebrospinal fluid was tapped an adapter connected with a rubber tube attached to an open 20 cc. syringe was inserted into the needle and about 10 cc. of the fluid allowed to run into the syringe. Ten drops of a solution of mercury bichloride containing 1/50 of a grain was then poured into the syringe. The tube was pinched and the syringe shaken: when the solution was well mixed and the froth settled down the fluid was allowed to flow back into the cistern by gravity. Within a few minutes the patient began to vomit and complain of headache, a reaction which usually lasted for one or two days.

In two cases treated by repeated injections after the above fashion arrest or definite improvement was obtained. Other authors have reported equally gratifying results by the same technique (Gifford, Keegan).

S. A. K. W.

[158] **The action of bulbocapnine in three cases of paralysis agitans and one case of tremor of paralysis agitans type.**—H. De Jong and W. Herman. *Arch. of Neurol. and Psychiat.*, 1926, xvi, 55.

Four patients, of whom three showed the typical Parkinsonian syndrome, and the fourth a tremor of the Parkinsonian type but no other signs, were treated with a group of drugs known to have a quieting action on the central nervous system. Of these four patients, two showed a striking amelioration of the tremor after bulbocapnine and two showed a questionable improvement. Two patients showed a striking improvement after scopolamine, and two were entirely refractory to scopolamine. All four were refractory to atropine and phenobarbital. Morphine was administered to only one patient, who had responded particularly well to bulbocapnine and scopolamine. It was found to have no effect on the tremors. Judging from the four patients examined, bulbocapnine and scopolamine alone can be regarded as of real therapeutic value. Atropine and phenobarbital are of no value. It is evident that cases refractory to both bulbocapnine and scopolamine are not rare. The cause of

this is not known. In the refractory cases, twice the dose given under ordinary conditions was administered, and though the patients were conspicuously drowsy, the tremor was unaffected.

R. M. S.

[159] **The relation of the cæcum-ascending colon content to idiopathic epilepsy.**—G. E. Armstrong. *Practitioner*, 1926, cxvii, 288.

The author reports on two cases of petit mal successfully treated by operation. He holds with Oppenheim that epilepsy is a toxipathic condition and with Collier that it is a "metabolic dyscrasia" and refers to cases with former brain injuries in which fits develop only as the result of taking alcohol. He considers that the most usual places from which toxins are absorbed are the cæcum and ascending colon.

In the first case, that of a man of 44, he removed the lower end of the ileum, the cæcum and the ascending colon, after which the fits ceased at once and have not recurred since 1921. In the second case, that of a girl of 11 years, the cæcum and ascending colon were removed: the fits ceased in ten days and have not recurred.

E. Casson.

Psychopathology.

PSYCHOLOGY.

[160] **The adjustments and unity of the organism.**—William A. White. *Psyche*, 1926, vii, 5.

Simple cells show a lack of organization. In living matter there is a tendency for paths of energy to become permanent and it is in this tendency that we find the beginning of *structuralization of function* or the formation of definite tracts by energy. These energy channels become permanently organized, the organization being transmitted to form part of the organism. Later, organization follows a definite plan and we find head and tail ends, etc. Experiments show that energy is highest at the point where the stimulus is received. The tapering of energy intensity is called the *dynamic gradient*. From these conclusions it is suggested that the single-celled organism has a head in effect, if not in structure, and the beginning of mind. Mind is the final representation of the function of the organism as a whole. In the single cell is found the commencement of psychological types of reaction just as are found the simplest types of structural organization. Having touched on the evolution of the nervous system the author expresses a belief that all the internal organs have reached the limit of their development but that the cerebral hemispheres may continue to increase in size and complexity. Mind is a function which has not yet become structuralized. It is suggested that bodily structures were originally mental. In the lower types function appears long before structure. We cannot lay down functions in structure until they have been well tried through the ages. Mind being function without structure is very adaptable. Automatic mental processes such as control the viscera and endocrine glands represent stages which are intermediate between this fluid quality of mind and definitely laid-down structures. Malignant growths are explained in terms of the organism's dynamic gradient, the controlling force at the head end being insufficient to affect all parts of the body equally: local irritation will thus develop an organization of its own. Instincts are divided into two main groups, the sex or life and ego or death instincts. In this connection no mention of Freud is found.

Robert M. Riggall.

[161] **An experimental study of mental and physical functions in the normal and hypnotic states.**—Paul Campbell Young. *Amer. Jour. of Psychol.*, 1926, xxxvii, 345.

On the whole there is no noticeable difference between normal and hypnotic states in the ability of normal persons in the fields of sensation, perception, fine discrimination, present memory (learning and retention), or physical work which does not involve fatigue. It is proved, however, that memory for long

past events is much better in hypnosis than in the waking state. Experimental results agree with the prevalent conception that rapport spontaneously appears in the deeper stages of hypnosis and that memory is greatly affected in the post-hypnotic period, but they are distinctly negative in regard to spontaneous catalepsy and complete posthypnotic amnesia as differentiæ of any grade of hypnosis. For the most part, hypnosis seems to consist in adopting with great conviction an attitude of mind. Even though all mental functions in the two states should be proved to be equal, still the differences in what the subject will and will not do, i.e., his ability to assume an attitude, are enough to differentiate the two states. This ability to play a part, so to speak, and yet be quite unconscious that he is playing it, may effect increased memory for long-past events in much the same way that, with some persons, throwing the body into a certain posture may enable them to recall incidents in connection with a certain man who habitually exhibited that posture. This taking of an attitude (consequently *refusing* to feel the pain or even to take cognizance of it) seems to be a description of the state of mind when analgesia or resistance to fatigue is induced in the hypnotic subject.

The basic difference, then, between the normal and the hypnotic state seems to be a difference, not in strictly mental or even physical abilities, but in the attitudes which can be assumed with verisimilitude. These hypnotic attitudes are emotional, impulsive, and voluntary rather than intellectual. "This would leave the term 'hypnosis' with a fairly definite meaning: a state in which a person *will do*, in a *bona fide* manner, possessed of conviction, what he *will not do* in waking life for lack of such conviction."

C. S. R.

[162] **Psychogalvanic studies on sixty-four medical students.**—Hans C. Syz. *Brit. Jour. of Psychol.*, 1926, xvii, 54.

Formal elements of the galvanic curve were singled out and linked up with concomitant characteristics of the emotional make-up. By this method the psychogalvanic reflex phenomenon may be used as an indicator of characteristic features of the emotional make-up of groups of individuals and in a lesser degree of single individuals. As stimuli were chosen: a list of fifty words, sensory stimuli, threats, deep breathing, and the suggestion to imagine a situation involving emotion. The average number of words reacted to was about one-third of the total list. The reactions were for the most part in close connection with the stimuli. Spontaneous waves occurred in only a few cases in which there was a high emotional sensitivity with a tendency to instability. The comparison of the frequency with the subjects' description of their own emotional make-up gave definite correspondence inasmuch as the individuals with smallest number of responses were of a well-balanced and not especially sensitive emotional constitution, whereas the cases with most frequent reaction were of a more excitable and labile emotional type. The shape of the galvanic

and respiratory curves was in these normal individuals preponderantly regular. Individuals with many galvanic reactions tended to show a low electrical resistance; but low resistance, on the other hand, was not found to be necessarily indicative of a high frequency of responses nor of special emotional characteristics. Variations of the resting current showed no consistent correlation with emotional traits of the individuals experimented on.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[163] **On Neuroses** (Zur Neurosenfrage).—Niessl v. Mayendorf. *Allg. Zeits. f. Psychiat.*, 1926, lxxxiii, 441.

The general grouping of neuroses into hysteria and neurasthenia is felt to be inadequate, and in view of the failure to establish any pathological anatomy of neuroses the problem needs tackling from another standpoint. On the other hand, the tendency to regard 'functional' as equivalent to 'psychogenic' leads us astray into the realm of mysticism. That emotional stress may have physical effects, and produce symptoms, no one can doubt; but proof is lacking that these can produce lasting nervous disease or psychosis.

It is clear that we must regard neuroses not as disease processes, but as pathological reactions of a morbid disposition based on inborn factors. The author believes that such predispositions may be classed in the two groups neuropathy and psychopathy, even though we find mixtures of the two groups of symptoms. From this foundation normal stresses elicit abnormal reactions.

In the neuropathic group he distinguishes four modes of reaction:—

(1) Painful reactions—headache, neuralgias, etc.

(2) Asthenia—lack of energy, fatigability, unrefreshing sleep, irritability, and the general reactions of neurasthenia.

(3) Neurotic organopathies—morbid reactions in internal organs and vasomotor system, nervous indigestion, etc.

(4) Spasmodic reactions—tics, twitchings, contractures, and hysterical fits, and in his view also idiopathic epilepsy.

In the psychopathic group he includes:—

(1) Paranoid reactions—a general tendency to suspicion, periodically increased by stresses.

(2) The hallucinatory reaction—without confusion and with insight except during the actual duration of the hallucination; closely linked with this is the tendency to hallucinate in response to suggestion, including the imagining of a suggested paralysis.

(3) Obsessional ideas and actions—phobias, perversions, drug habits.

(4) Transitory states of depression other than manic-depressive.

(5) Abnormal characters, pathological liars, etc.

He believes that treatment should first aim at lessening or removing the disposition to morbid reaction to stress; he has found that psychotherapy may often remove the present symptom, but increase the morbid disposition. Since the individual cannot adapt himself to all conditions, treatment must consist largely in finding out those conditions in which the least adaptation is required and adapting his environment to his needs. For prevention this should begin in childhood.

M. R. BARKAS.

[164] **The physico-psycho-galvanic reflex in the neuroses and psychoses.**—P. K. MCCOWAN. *Jour. of Ment. Sci.*, 1926, lxxii, 492.

THE galvanic reflex is an inevitable response, and alone of all such responses which can be studied objectively, it is specific to 'nocuous' stimuli. It thus becomes a most important objective method of investigation into the neuroses. Reaction-times as obtained in word-association tests can be exceedingly misleading as 'complex' indicators, but taken in conjunction with the galvanic reflex, simultaneously obtained, can give valuable information. A study of this reflex suggests that the various neuroses are not definite entities but merely clinical syndromes. Three types of affectivity which cannot be diagnosed clinically with any certainty are found in the neuroses: (*a*) increased affectivity: (*b*) diminished affectivity: (*c*) affectivity, otherwise normal, increased to special complexes. The large majority of hysterics have diminished affectivity, and it is suggested that their symptoms are due to the failure of their diminished emotivity to act as the mainspring of their volition. In anxiety neurotics increased emotivity is usually present, but there are many exceptions to this rule, and in the opinion of the writer these exceptions are very closely allied to, and might well be classed with, the hysterics. The symptoms of these patients with increased emotivity is due to their exaggerated emotional reactions making them attach undue importance to their experiences in accordance with their excessive emotions. The affectivity varies in different cases of dementia præcox, but in his series all hebephrenics showed increased affectivity.

C. S. R.

[165] **The pathogenesis and significance of cyclothymia** (Zur Pathogenese und Bedeutung der Cyclothymie).—P. PRENGOWSKI. *Allg. Zeits. f. Psychiat.*, 1926, lxxxiii, 491.

IN this interesting paper the author describes three cases in which alterations of emotional state, motor and intellectual activity, and digestive disturbances appeared to follow changes in cutaneous vascular and secretory mechanisms induced by changes of temperature, to which the patients reacted with abnormal sensitiveness. Full details of the cases are given. In each the initial morbid condition appears to be a spasm of the skin blood-vessels, the skin becoming

cold and pale, the lips blue, sweating greatly diminished, dermographia marked; correction of this by a superheated atmosphere, hot baths, or drugs dilating the skin vessels results in removal of the inability to think, concentrate, and work, as well, in at least one case, of constipation.

He discusses the possible ways in which the physical and mental reactions may be connected: it is uncertain whether the cerebral blood-vessels are affected by the same state of spasm, or whether they undergo compensatory dilatation, or whether the two states occur in vessels of different distribution. He quotes experiments in rabbits showing that cold applied to the skin vessels caused dilatation of those of the pia mater, and quotes various authors who associate cerebral vascular changes of a functional kind with abnormal mental states. Another possibility is that the reduced secretion of sweat causes the retention of toxic substances, and another that circulatory changes in the brain lead to the accumulation of fatigue products.

He discusses the diagnosis of these cases, which come rather into the group of periodic neurasthenia than into that of manic-depressive psychosis; further, certain of the manifestations—apathy, mannerisms, stiffness, and a tendency to progressive emotional loss—resemble dementia præcox, in which too circulatory disturbances are marked.

This view of cyclothymia leads him to certain conclusions as to therapeutic indications, which he intends to publish later.

M. R. Barkas.

PSYCHOSES.

[166] **Some symptoms referable to the basal ganglia occurring in dementia præcox and epidemic encephalitis.**—C. Farran-Ridge. *Jour. of Ment. Sci.*, 1926, lxxii, 513.

Among all the psychoses whose symptoms may be simulated by epidemic encephalitis, dementia præcox occupies the foremost place; in fact the semiological resemblance between the two diseases is in some cases so striking as to give rise to real difficulty in differential diagnosis. It is suggested that this special resemblance is accounted for by the fact that just as in epidemic encephalitis the virus tends to attack selectively, or at least predominantly, the basal ganglia, so in dementia præcox, at any rate in the katatonic variety, the main incidence of the disease process tends to fall on the same portion of the brain. In dementia præcox in the acute stage there is usually a very considerable fall in the body-weight and later flesh is put on in an extraordinary way. Similar changes occur in general paralysis and in epidemic encephalitis. There are many symptoms which can be referred with a high degree of probability to a disturbance of the hypothalamic sympathetic centres, and it seems likely that these disorders of metabolism are nervous rather than glandular in origin, although the pituitary body may be the mechanism through which the

hypothalamus acts. Blepharoclonus and allied phenomena are noted in dementia præcox and encephalitis. The absence in cases of dementia præcox of any evidence of nuclear involvement of the external eye muscles tends to exclude the mesencephalon, while the association of trembling of the eyelids with Parkinsonism and with stupor, and the occurrence of the same symptom in paralysis agitans and hysteria, are all facts which speak in favour of the localization of the lesion in the basal ganglia. Pruriginous phenomena are, too, found in dements, cases of general paralysis and encephalitis, and the author thinks these are abnormal sensations of thalamic origin. This is true also of chewing movements and teeth-grinding found in similar states. 'Greasy face' also occurs in dementia præcox and its association with Parkinsonism, with stupor and flushing of the face, tends to show that the hypersecretion of the sebaceous glands is in some way connected with disturbance of the sympathetic centres in the basal ganglia. Various respiratory disorders are also common to these conditions. In the present state of our knowledge any discussion as to the pathogenesis must be largely speculative. In favour of the causal lesions being situated in the basal ganglia we have (1) the frequent association of respiratory abnormalities with Parkinsonism and with inversion of the sleep rhythm; (2) the absence of co-existing bulbar or pontine symptoms in dementia præcox and the rarity of such symptoms in epidemic encephalitis. The occurrence of similar disorders in hysteria seems to speak in favour of their thalamic origin. Choreiform movements and 'tachykinesia' (actions of extraordinary abruptness) are brought forward as confirmatory. The author states that he does not regard the choreiform manifestations of the dement as motor functions of the corpus striatum, for it is obvious that they are cerebral in type. Nevertheless it is considered that the basal ganglia are concerned in their production and Kinnier Wilson's view is adopted that although choreiform movements are exteriorized *via* the corticospinal paths, behind their appearance is an afferent disorder of regulation attributable to lesions situated on the cerebello-mesencephalo-thalamo-cortical path.

C. S. R.

[167] **Frequency of dementia præcox in relation to sex, age, environment, nativity and race.**—Horatio M. Pollock. *Mental Hygiene*, 1926, x, 596.

In the United States it is found that dementia præcox occurs more frequently, and earlier, among males than among females. It is more prevalent in cities than in rural districts. It is more prevalent among the foreign-born than among the native population in both city and country. It is more frequent relatively in some European races than in others. It is more prevalent among negroes than among whites.

C. S. R.

[168] **Blood-vascular conditions in psychoses.** — Isabella McDougall Robertson. *Jour. of Ment. Sci.*, 1926, lxii, 356.

In a previous communication it was shown that the hæmoclastic crisis does not occur in the normal healthy subject, but that it does occur in a large percentage of psychotics. Analysis of a group of 90 well-established cases of mental disorder showed that 94 per cent. were schizophrenic in type, 85 per cent. were cases of melancholia, and 75 per cent. chronic mania. In 260 early psychotic and neurotic cases, a hæmoclastic crisis was found in over 60 per cent.; it was noted that the greater number of these were psychotic, chiefly of the schizophrenic type, and that most of the neuroses included in this 60 per cent. were anxiety forms. Consideration of the subsequent progress of 148 of these cases showed that 55 per cent. of the positive cases were reported as worse, 24 per cent. were better, and that 70 per cent. of the negative cases were reported as better and only ten worse. Consideration of later progress during a further period showed that there was very little change in the proportion of cases included under the different headings. The writer's observations are in agreement with those who consider that the cause of the hæmoclastic crisis is a change in the equilibrium between vagus and sympathetic tonus. An abnormal response to postural change was found in 87 per cent. of the psychotics. This response was purely a vasodilation.

C. S. R.

PSYCHOPATHOLOGY.

[169] **Syphilis and mental disease.**—H. Ferguson Watson. *Jour. of Ment. Sci.*, 1926, lxxii, 573.

The object of this paper is to record the results of systematic serum tests. The investigation was carried out in the Manx Mental Hospital, where 290 cases were studied, and in the Argyll and Bute Mental Hospital, where 388 were studied. These may be regarded as collectively representative of the general types of mental disease. In the first-named, 46 cases gave positive results—23 men and 23 women; 241 were negative—117 men and 124 women; while the cases of one man and two women were positive with the flocculation test and negative with the Wassermann reaction. The male patients gave positive results in 16.31 per cent. and the female in 15.43 per cent. Among those in the Argyll Hospital, of the 185 men and 203 women, 39 of the former and 33 of the latter gave positive results: 139 men and 169 women gave negative results, while the case of one woman was positive with the flocculation test and negative with the Wassermann reaction. The same result was obtained when these cases were tested a month later. The male patients gave positive results in 21.08 per cent. and the women in 16.25 per cent. In the series of cases studied the author concluded that the flocculation test, if not superior to the Wassermann reaction, has been proved to be in no way inferior, and its application is much

simpler. The investigation suggests that while syphilis in mental hospitals is more common among men than among women, there is not such a wide difference as was at one time supposed. By means of laboratory methods it can be demonstrated that the incidence of syphilitic infection is much greater than had been suspected. The percentage has been raised by 12.1 in the case of the Manx patients and by 15 for the Scottish Hospital. Better conclusions can be arrived at when a greater number of mental hospitals are available for comparison.

C. R. S.

[170] **The Meinicke micro-reaction for syphilis for use in a mental hospital.**—R. M. CLARK. *Jour. of Ment. Sci.*, 1926, lxxii, 588.

THIS is the Meinicke turbidity reaction for the serodiagnosis of syphilis improved and adapted to a microscopic slide test ; venous puncture is unnecessary, for sufficient serum is got from a drop of blood collected as for a Widal reaction. In 1925 Meinicke improved and simplified Dohnal's method, and after a comparison with the Wasserman reaction in 2,000 sera, satisfied himself that the methods were of equal value for the detection of syphilis. This reaction has attracted little attention in this country, although it is extensively used in European laboratories. The writer concludes that it is a valuable addition to laboratory methods for the serodiagnosis of syphilis. It is simple, ready, and accurate, and will be found especially useful in the case of the insane. Large numbers can be dealt with in a short time. It is an excellent test for use in a mental hospital and for the routine examination of admissions.

C. S. R.

[171] **Syndromes of preadolescent mental regression** (Sindromi di regressione mentale infanto-giovanile).—G. CORBERI. *Riv. di pat. nerv. e ment.*, 1926, xxxi, 6.

THE author describes four cases in full and refers to other conditions which he thinks should be separated from the general group of idiocies under the above title. They are characterised by a diffuse morbid process in the central nervous system, marked firstly by a lipoidal neurolysis and secondly by sclerosis.

He suspects congenital syphilis as an important pathogenic factor. In some cases a febrile condition is observed at the beginning of the syndrome but its real nature cannot yet be properly interpreted.

R. G. G.

[172] **Suggestions on the psychology of mental deficiency.**—HUBERT C. BRISTOWE. *Jour. of Ment. Sci.*, 1926, lxxii, 364.

AFTER a discussion of instinct and intellect in the lower animals the writer attempts to apply these views to the mentally deficient. Mental deficients may be divided into two main classes, moral, and intellectual. It does not follow that a moral deficient is wanting intellectually, or that the intellectual

deficient is lacking in moral qualities. The two forms cannot have a common origin and they are regarded as due to an absence or deficiency of one or other of the two primary instincts, racial, and egoistic. There are many cases of moral deficients who are well able to earn their own living and even possess a high standard of intelligence, who in all other respects are normal, but the life and comfort of others are immaterial to them so long as they can satisfy their own desires. The egoistic instinct, no longer held in check by the altruistic, runs riot. The racial instinct is the parent of all morality and morality is in itself that instinctive guidance which is necessary for the well-being and continuance of the race. The problem of intellectual deficiency is very much the same when we have excluded cases of gross physical maldevelopment; it appears that the egoistic instinct has been partially or entirely interfered with in its transmission from the parent stock. If we look back on the qualities which go to build up intellect, we find that the lower the mental grade the more of these qualities are lacking. Even in the higher grades we find that peculiarly human quality, the power of grasping an abstract idea, is almost always lacking. One cannot trace in the mentally deficient that inhibitory power which is almost synonymous with wisdom. Between the degraded statutory idiot and the high grade deficients we get every type. The lowest is incapable of taking the smallest care of himself, and lacks every instinct which is common to the human being and even to the lower animals. As we descend from the highest grades of deficiency, we first notice the lack of reasoning power, i.e., the lack of the power of inhibition; lower down the scale we find an incapacity to grasp abstract ideas, and then curiosity becomes more or less wanting and finally all power of self-protection. Moral deficiency is, as one would expect, always present in the lowest mental degenerates. To sum up, morality develops from the racial instinct, and perversion or absence of that instinct leads naturally to the perversion or absence of morality. Intellect develops from the egoistic instinct, and perversion or absence of that instinct is the cause of want of growth of the intellect.

C. S. R.

[173] **The prevention of insanity: a preliminary survey of the problem** (The seventh Maudsley lecture).—GEORGE M. ROBERTSON. *Jour. of Ment. Sci.*, 1926, lxxii, 454.

MUCH statistical work with charts is given us. Insanity under the age of 15 is quite negligible. A fair number of admissions occur between 15 and 19, and a larger number between 20 and 24. The amount continues up to 40 years of age, after which it falls. This represents the number of cases of insanity occurring at the different ages, but not the liability to insanity. The large number of admissions recorded between the ages of 20 and 40 is mainly due to the greater number of young persons in the community, and the small number alive at that age. The tendency to develop insanity first shows itself between

15 and 19, then becomes much more pronounced and is followed by a steady rise till the age of 50 is reached; then comes a short period of decline, after which an extremely rapid rise occurs in old age. There is less insanity at adolescence than at any subsequent period and a general tendency for insanity to become more frequent as one grows older. The rate among males is proportionately higher than among females, mainly due to the greater number of admissions from general paralysis and alcoholic insanity. Incidence among the married is decidedly low. As regards widows and widowers, it is found that the loss of a husband or a wife increases the incidence of insanity in a marked degree. Prevalence among the single is decidedly high. At the three crises of life (adolescence, climacteric, and the grand climacteric at 64) we note an increased incidence. Dementia præcox is most prevalent in early adult life and as in the population young persons greatly predominate, the number of admissions is large. Since the majority do not recover, and fill our mental hospitals, the impression has thus been created that the early period of life is the most liable to insanity; whereas if we refer to insanity in general, adolescence is actually the least dangerous period. Manic-depressive forms by far the largest proportion of re-admissions (30 per cent. males, 48 per cent. females). Dementia præcox constitutes 25 per cent. Alcoholic insanity and general paralysis account for more than a half of all the insanity that occurs between the ages of 35 and 55. The highest point of the incidence of the former is between 45 and 49, and this would seem to indicate that there is in the male a climacteric which corresponds to the female menopause. General paralysis begins later, terminates sooner, and reaches its period of maximum incidence five years earlier than is the case with alcohol.

In the latter part of the paper the various factors of prevention are discussed.

C. S. R.

PROGNOSIS AND TREATMENT.

[174] **A note on the Boltz test in the cerebrospinal fluid after malarial therapy in general paralysis.**—G. W. T. H. Fleming. *Jour. of Ment. Sci.*, 1926, lxxii, 376.

Boltz found this test almost invariably positive in general paralysis and negative in the other psychoses. The test is not due to cholesterol by itself, but would appear to be due either to some combination of cholesterol with protein or some modification of cholesterol. At the Sunderland Mental Hospital 13 cases of general paralysis were treated with induced malaria by the subcutaneous route. The malarial blood was obtained from a general paralytic and was injected ten hours after its withdrawal. Of these 13 cases, three died before the cerebrospinal fluid was examined, and of the remaining ten, of which nine had a positive Boltz test previously, nine had a negative test

after the malarial treatment. One case whose Boltz test was negative previous to treatment, became positive after treatment. In these nine cases the change from a positive to a negative Boltz test suggests that as a result of the malarial therapy, apart from any obvious clinical and serological improvement, the paretic process which is responsible for the substance causing the Boltz test had become inactive. Whether this improvement will be permanent or not remains to be seen.

C. S. R.

[175] **The value of malarial therapy in dementia paralytica: preliminary report from the London County Mental Hospitals Service.**—*Brit. Med. Jour.*, 1926, ii, 603.

MALARIAL therapy produces a greater number of remissions and a smaller death-rate than occurs in untreated cases. Complete remissions occur in about one-third of the cases in a mixed series, but in a much greater proportion if only cases with histories of dementia paralytica of less than eighteen months' standing are treated. The addition of neosalvarsan to the febrile treatment decreases the death-rate amongst the cases unfit for discharge, and perhaps increases the number of complete remissions that occur.

All the medical officers are agreed that in a majority of the cases treated by malarial therapy which proved to be unfit for discharge a marked improvement in general well-being, cleanliness, and conduct was observed.

C. S. R.

[176] **An investigation into the chemiotherapy of general paralysis** (Recherches sur la chimiothérapie de la paralysie générale).—SEZARY and BARBE. *L'Encéphale*, 1926, xxi, 1.

THIS is the record of an attempt to find some drug or combination of drugs which would give definite results in general paralysis. Mercury was ruled out, as the authors considered it to have a definitely maleficent action on the disease. The drugs dealt with in this report are the arsenicals (eparseno, arsacetine and tryparsamide) and the bismuth preparations (trepol, quinby, rubyl, iodo-bismuthate of pyramidon, luatol and the bismuthogallate of ethylene diamine). These were in some cases combined with other remedies such as antidiphtheritic serum, mesothorium, sodium nucleinate, hemostyl, cerebrocrinol and gardenal. The authors found that bismuth salts were quite without effect on the course of the disease. Of the arsenicals used, eparseno was comparatively useless, and arsacetine was apt to cause the rapid development of optic atrophy. Tryparsamide was only used on three patients, one of whom was apparently cured, one remained stationary, and one died. One patient treated with 31 injections of dinitrophenol improved remarkably and remained well. The authors claim to have had good results with stovarsol but these are not included in the present article.

J. G. GREENFIELD.

[177] **Sterilization of mental defectives.**—*Jour. of Ment. Sci.*, 1926, lxxii, 386.

Few people who have not made a study of the subject realize to what extent the sterilization of criminals and mental defectives has been sanctioned by the legislatures of America. A recent case which upheld the main provisions of the present Michigan statute authorizing the sterilization of mentally defective persons, is one indication of what seems to be a growing popular approval of such statutes. Laws providing for sterilization in specified cases have been placed on the statute books of twenty-two states. The legislature of other states has considered the subject, and at least two of them have passed laws which were vetoed. Such laws in Indiana, Iowa, Michigan, Nevada, New Jersey and New York have been declared unconstitutional. Iowa and Michigan have since enacted new laws. The New York law of 1912 was repealed in 1920. The latest laws of the other states have not yet been tested in the courts.

Enforced sterilization has been practised by many peoples, usually as a punishment for sexual offences, but it is only within the last twenty years that it has been given serious consideration by state legislatures as a eugenic measure. Some state statutes are purely punitive but the great majority are eugenic and therapeutic, or purely eugenic. Those that are punitive of course apply only to criminals, usually confined to those who have been convicted three or more times of a felony, or to those guilty of rape or other sexual crimes. Mostly these laws apply only to inmates of state institutions. Authority is given to sterilize such inmates when in the opinion of the examining board procreation is inadvisable because the children would have inherited tendencies to crime, feeble-mindedness, insanity, etc. A few statutes include those who have syphilitic disease, and some include sexual perverts.

The majority of the statutes provide for the operation of vasectomy on males and salpingectomy on females. Some leave the method to the discretion of the examining board. Some allow castration and some specificially prohibit it. The present Michigan statute is the only one to speak of treatment by *X*-ray. Under most of the laws no consent of parents or guardians is necessary. Most boards are assisted by a certain number of physicians and neurologists. Recent statistics are not available, but up to March 1918 the number of operations performed under these statutes was: California 1,077, Connecticut 12, Indiana 118, Iowa 67, Kansas 3, Oregon 17, Nebraska 25, New York 9, North Dakota 32, Washington 1, Wisconsin 61, other states 0. From 1907 to 1921 in California 2,588 persons were sterilized, and during the same period in the various states a total of 3,233 were sterilized.

The advisability of these statutes has been seriously questioned. Since the majority of them are eugenic they are necessarily based upon two assumptions: (1) that feeble-mindedness, insanity and criminal tendencies are inheritable; (2) that it is possible to determine in a particular case that children procreated by a certain defective will have such inherited tendencies. These

are assumptions about which there is difference of opinion. Though it has been stated that "biological science has definitely demonstrated that feeble-mindedness is hereditary," on the other hand it has been said that "there has always been some uncertainty in making a diagnostic distinction between native feeble-mindedness on the one hand and the acquired defect resulting in retardation on the other." It is contended, too, that these laws open the door to other and greater evils.

C. S. R.

[178] **Psychiatry and legal detention** (Psychiatrie und Bewahrungsgesetz).—Mönkemöller. *Allg. Zeits. f. Psychiat.*, 1926, lxxxiii, 448.

In this lengthy paper the author discusses the whole problem of the social and psychopathic individuals who are a burden and often a danger to society, and who require some sort of segregation and treatment. For those who are definitely mentally defective, he urges the prolongation of their retention in institutions beyond the usual age, up to at least about 26, when they no longer have puberty stresses to contend with in addition to their general unfitness for a normal social environment. For the habitual criminal he urges lengthy detention with liberation only on ticket-of-leave, so that the ease of returning them to the institution may provide a motive for the abandonment of a social conduct.

He points out the disadvantages of repeated short convictions for petty crimes in the feebleminded and morally abnormal, and suggests that existing institutions might be differently utilised and some new ones founded. The workhouses might be the first place to which the psychopathic asocial should be remanded, but in these occupational therapy of a varied kind is needed and psychiatric supervision, so that re-education to work and self-maintenance may be furthered. When this fails to be effective after several shorter periods with relapses, then places of detention for longer periods, probably with an initial commitment of three years, to be extended if need be, should be set up, where conditions are something like those of mental hospitals, definitely not prisons or for punishment, but for treatment of the asocial tendencies and for re-education. Many passive types of asocial persons, simply incapable of independent existence outside, would work well under supervision and willingly remain in this protected environment. The actively asocial would probably require stricter discipline with rewards and penalties of some kind.

The main difficulty would seem to lie there as here in the fact that the public dreads further restrictions of the liberty of the subject, and the desirability of segregation for long periods and even permanently of the high grade feeble-minded and moral defective, the psychopathic characters with defects of will and emotional control, is not readily appreciated by the judicial authorities in whose hands the commitment must lie. He suggests that some place of observation and provisional detention, with welfare workers gathering full

information of the past conduct of the patients and psychiatrists specialising in such work, might bring cogent evidence of the advisability of segregation and treatment. He urges that psychiatrists should claim such cases as belonging to their field of work, and even where asocial and antisocial tendencies are incurable, that they can most advantageously be kept occupied and supervised by a staff with the psychiatric rather than the prison point of view.

M. R. BARKAS.

[179] **Divergent tendencies in psychotherapy.**—JAMES GLOVER. *Brit. Jour. Med. Psychol.*, 1926, vi, 93.

THIS paper deals with certain broad issues dividing those practising psychotherapy, the most fundamental of which depend on its conception as an applied science. Discussing certain grounds for scepticism and half-hearted reliance on psychological techniques, the author comments on Reik's suggestion that a motive for psychologizing might be due to a desire to satisfy curiosity without uncovering the deeper determinants of our mental processes. He thinks that this reluctance is not entirely due to any physiological bias in medical men but may actually constitute a psychophobia. Emphasis on physical aspects of neurotic illnesses is probably determined by some inhibition connected with the exploration of their psychological causation. The psychotherapist tends to be influenced by a physiological bias which causes him to overestimate environmental influences and underestimate the importance of inner forces. It has recently been demonstrated that the fate of *inner* instinctual 'urges' plays a far more important part in the etiology of neuroses than environmental factors prevailing at the onset of the illness. The author believes that man's capacity for achieving profound modification of instinct is now in part inherited. Neurosis is the expression of failure to establish a compromise between certain instinctual claims and the counter claims of a cultural self created in the image of the parental representative of social ideals. The repression of instinctual demands and the failure of the neurotic satisfactorily to *displace* these strivings on socially acceptable ends, make it impossible to envisage the patient's relation to his environment as a simple biological one such as is established by animals in captivity. We are thus unable to build Mappin Terraces of our neurotic patients. The neurotic's inability to maintain a harmonious relationship to his environment is only partly due to the frustration of conscious aspirations; these factors can only precipitate the onset of neurosis. The treatment of neurotic disorders by modification of environment is considered and is shown to be a 'hit-or-miss' therapy; it can only be a palliative measure. Neurosis can only be stated in biological terms of 'organism reacting to environment' when we take into account the modification of instinct by the cultural *milieu* which takes place at the beginning of life. Scientific therapy should aim at better adjustment of internal conflicting forces rather than at reaction to environment. The view that a scientific psychotherapy

requires to be supplemented by philosophic or religious doctrines is criticised. It is possible to question the scientific validity of this standpoint without denying that religious beliefs help the individual in time of stress. In his criticism of ' medical moralization ' it is noted that the neurotic is not so much concerned with ordinary temptations as with his arrested infantile cravings. Medical moralization will thus leave the real source of guilt untouched and may dangerously increase the already too strenuous activities of the super-ego. The paper concludes with a lucid account of the mechanism of transference and how it can be applied either to influence the patient directly or as a means of reviving memories of his forgotten infantile past. It cannot be used along both of these divergent directions. Analysis automatically achieves synthesis. To be scientific the transference situation must be conducted according to the rules of psychoanalysis.

ROBERT M. RIGGALL.

Reviews and Notices of Books.

Epilepsy, a Functional Mental Illness: its Treatment. By R. G. Rowe, M.D. Lond., Pathologist and Medical Officer, County Mental Hospital, Prestwich, Manchester, and W. E. Bond, M.R.C.S., L.R.C.P., a Senior Medical Officer to the Ministry of Pensions. London: H. K. Lewis & Co. 1926. Pp. 138. Price 8s. net.

Any serious attempt at a solution of the numerous and complex problems offered by epilepsy deserves consideration, for they must be obvious to any observer other than the superficial. The rationale of treatment should be the outcome of exact knowledge of its etiology and pathogenesis, but such knowledge is still to seek. The late Dr. R. G. Rows busied himself greatly with the question of epilepsy as it occurred in soldiers during the war, and relied very largely upon military cases in endeavouring to find a common basis for all kinds of epileptic manifestations. His collaborator, Dr. W. E. Bond, has published the results of their investigations just as the record was written by Dr. Rows, indicating in a brief preface that he was disinclined to alter anything, even if theories of his partner are "open to question, or since disproved." The reviewer is thus rather under the impression that the book cannot perhaps be taken to represent finality in respect of its authors' views and, if so, criticism is to some extent disarmed.

Be this as it may, the conclusion herein reached is to the effect that in every type of case the one common factor is "a disturbance of consciousness with which is associated an emotional state." The reception of a stimulus from the outer world or from some organ of the body "leads to the revival of a memory and the emotional state associated with it." Hence there is "fixation of the attention," the consequence being movements of head, eyes, pupils, and other parts of the body "usually associated with the processes of attention," and also movements of the limbs or organs of the body "by means of which the emotions are expressed." The aura is a recalled memory, and if the reaction spreads beyond the stage of an "intense emotional state and increased concentration of attention" this occurs "through the extra-pyramidal paths which proceed from the cortex to the subcortical ganglia," (sic). The 'cry' of the epileptic patient "frequently suggests a condition of extreme fear": in such cases the motor reactions are movements of the head, limbs, body (and of internal organs), "which are usually employed to express the emotions."

This courageous attempt to unify the processes of epilepsy, whatever the clinical type, whatever the actual existing condition (cerebral tumour or shell shock), whatever the degree and duration of the phenomena, would

perhaps be more plausible were it not based to some extent on anatomo-physiological considerations which are open to obvious objections. The authors rely on the presence of vasomotor nerves on the vessels of the central nervous system as being "definitely proved," whereas the recent researches of Florey render their existence more problematical than ever, and Leonard Hill has shown that if they exist their power must be entirely insignificant. For the exteriorisation of the centrally produced movements anatomical paths are postulated the existence of which is unproven, and old experiments of Bechterew and others are relied on which are capable of other explanations than those accepted for their theory by the authors. But apart from these and analogous difficulties to which no reference can here be made, it will we think be admitted that the crux of the matter, or at any rate one of the cruxes, resides in the problem of whether the movements of epilepsy have any 'meaning' or not. The authors consider the epileptic 'cry' often one of fear, but many who have heard the peculiar inspiratory rush of air through the larynx as the diaphragm spasmodically descends will not accept this interpretation. They believe that the writhing convulsions of the epileptic express emotion, and that the rigidity of head and eyes expresses attention. This view of the 'meaning' of the contortions of the attack we cannot entertain, and consider that it is belied by any objective examination of what actually takes in a fit. It is a curious thing that while Hughlings Jackson is extensively quoted, and with approval, there appears to be no citation of his opinion that in the convulsion the individuality of each movement is lost, that it is nothing else than "a single big useless movement," and that subsequent (clonic) movements "do nothing but mark time." That the phenomena of Jacksonian epilepsy are the expression of recalled memories of intense emotional states and of increase of attention seems to us just a little absurd. Of what emotion is clonic convulsion limited to the forefinger and thumb of one hand the expression? Nor can the clinical picture of many examples of petit mal be made to accord comfortably with these views. The authors have set out to prove too much and are consequently in difficulties when their theory is applied widely.

S. A. K. W.

Experimental Investigations into the Emotional Life of the Child, compared with that of the Adult. By H. Eng. Translated by George H. Morrison, M.B. With 9 plates. Oxford Medical Publications. Humphrey Milford: Oxford University Press. 1926. Pp. 243. Price 21s. net.

Laboratory experiments were undertaken by means of the plethysmograph to investigate the emotional life of the child between the ages of seven and fourteen. It was sought to determine whether psychical experiences of a definite character are accompanied by definite changes in the volume pulse curve and in the respiratory curve, and if the changes in these curves which

are found in adults are also found in children. Some 25 persons (11 children and 14 adults) were selected for the experiments.

The factors recorded were rise or fall of volume curve, increase or decrease of pulse height, and acceleration or retardation of pulse and respiration respectively.

It was found that attention and psychical work are not identical but give diametrically opposite records, but since they frequently occur together the results are obscured. Displeasure and pleasure, on the whole, show opposite types of reading, and intellectual and æsthetic emotions show certain special features. As to the difference between children and adults it is shown that spontaneous displeasure curves occur less frequently with children than with adults. Spontaneous displeasure curves are, in the case of children, mostly related to sensory impressions or concepts of sensory impressions. In the case of adults they are more often connected with more important events of greater personal psychical content.

Spontaneous displeasure emotion does not last very long, as a rule, in children, while in adults it is often of long duration. The phenomena of spontaneous displeasure emotion are never so strongly marked in children as they are occasionally in adults. This indicates that as a rule the spontaneous displeasure emotions of children are weaker.

Further, the spontaneous pleasure emotions of the child are bound up predominantly with sensory impressions, while those of the adult are more often connected with personal experience or with definite psychical processes. A relatively greater number of spontaneous pleasure curves are obtained from children than from adults. The pleasure emotion is apparently more easily aroused in children. Spontaneous pleasure emotions of children have longer duration than spontaneous displeasure emotions. On the whole, there is in the child a more marked spontaneous tendency to rising curves, and also a greater inclination to psychical activity of a pleasurable nature.

It is noticeable that all displeasure-producing stimuli do not produce the same results, nor do all pleasure-producing stimuli; therefore the author concludes that his experiments disprove the pleasure-displeasure theory of emotion. Nor does she find Wundt's three-dimensional theory more acceptable. From her experiments she concludes that the following six affective reactions exist, all giving different results with the plethysmograph: Attention emotion, Activity emotion, Displeasure, Pleasure, Depression and Excitement. She also states that her experiments disprove the James-Lange theory and show that matter depends upon mind and not mind upon matter.

R. G. GORDON.

Aphasia. By S. A. Kinnier Wilson. Psyche Miniatures (Medical Series), No. 2. London: Kegan Paul, Trench, Trubner & Co. 1926. Pp. 108. Price 2s. 6d.

It is a difficult enterprise to undertake, within restricted limits, an examination of the enormous problem of aphasia without the omission of any of the essential viewpoints from which that controversial question may be approached. This attempt, none the less, the author has made, and, we may add, with complete success. From the introduction we may gather some of his general contentions, such as the following: aphasia is not a disease, but a symptom; the function of language is an intellectual function, in regard to which the idea of localisation cannot have the spatial significance that belongs to a morphological unit. The author distinguishes thought from language, and follows Hughlings Jackson in maintaining a double symptomatology, negative and positive, for the disorders improperly termed aphasic (if the etymology of the word is considered).

A given aphasic symptom may be envisaged from three points of view: (1) What is the anatomical site of the lesion producing the defect? (2) What are the physiological mechanisms involved? (3) What is the nature of the psychological disorder? No study of the subject can make any pretence at completeness if any of these three aspects is ignored. The first three chapters are devoted to their study. Anatomical considerations can obviously take up only a small part of so restricted a volume, but the essential facts are enumerated. In his physiological considerations the author specially stresses the kinæsthetic element in speech. The psychological considerations include a criticism of the views of Pierre Marie and Moutier, to the effect that in the aphasia of Wernicke there is a combination of a general intellectual defect and a special intellectual defect of speech. A study of intellectual and emotional speech follows, in which connexion the author recalls his very interesting case of apraxia (published in 1908) in which, the lesion being in the right hemisphere, emotional speech was lost and intellectual speech conserved.

Modern observation compels us to abandon the idea of the destruction of engrams or images as being at the basis of aphasic syndromes, and to substitute for it the view that in the light of our present knowledge no adequate use can be made of these conceptions. The last three chapters are concerned with the classification of aphasic disorders, certain clinical types of aphasia, and its treatment. We note the scepticism of the author in regard to classification, in which respect he follows Jacksonian tradition. In no domain of knowledge is it more necessary to be at once neurologist and psychologist, and, we may add, linguist.

It would doubtless be easy enough to indicate omissions in this short monograph, though the criticism is scarcely fair. One would have liked, for instance, an exposition of the theory of chronogenic localisation associated with the name of von Monakow but derived from Jacksonian conceptions. Such as it is, this little book forms an excellent introduction, and the author's mastery of his subject evokes our admiration. R. Mourgue.

Das Augenzittern als Gehirnstrahlung ; ein Atlas der Augenzitternkurven. By Professor Dr. JOHANNES OHM, Bottorp (Westphalia). With 226 illustrations in the text. Berlin and Vienna : Urban and Schwarzenberg. 1926. Pp. 326. Price M21 ; bound, M24.

PROFESSOR OHM has had wide experience of miners' nystagmus among the coalgetters of Westphalia, and has made many previous contributions to that vexed question. The present volume deals more ambitiously with the whole problem of nystagmus. An amazingly elaborate analysis of the technical features of nystagmus, various ingenious instruments being utilised for the purpose, is followed by a description of the clinical features of the following varieties : miners' nystagmus, spasmus nutans, congenital amblyopic nystagmus, acquired amblyopic nystagmus, so-called railway nystagmus, vestibular nystagmus, nystagmus of central origin (Deiters nystagmus), and hysterical nystagmus. The third section of the volume is taken up with a minute examination of the theoretical side of the subject, the relation of the phenomenon to disorders of the optic and vestibular mechanisms being discussed in a painstaking way. At the end an eight-page bibliography is furnished.

It is a little difficult to summarise the author's views and conclusions, derived from study of an immense amount of clinical material, among which the reader is possibly a little apt to lose his way. Briefly, Professor Ohm holds that miners' nystagmus is an oscillatory phenomenon which is caused by interference with the frequency and strength of the excitations normally passing from the optic centres in the brain to the eye muscles ; it is a disorder of central innervation. To it the spasmus nutans of children is closely allied. Vestibular nystagmus is a reflex reaction whose arc does not extend higher than Deiters' nucleus ; optic nystagmus is a reflex phenomenon the arc of which includes the cortex. It is not easy, further, to follow the author in his comparisons of this physiological disturbance with the tone-variations of music and with the phenomena of wireless. When he declares that as the sun's energy radiates from coal so does a stream of living substance flow from certain cerebral ganglia to exteriorise itself in the movements of nystagmus we feel the language of simile is being unduly strained, as also when he says that through nystagmus the spirit of the ' physical ' makes a broad inroad on the ' neurophysiological.' The book closes with a ' Siegesgesang ' from Beethoven's Fifth Symphony.

S. A. K. W.

Handbuch der Neurologie des Ohres. Edited by Professor Dr. G. ALEXANDER and Professor Dr. O. MARBURG, Vienna. Volume III. With 200 illustrations, some in colours. Berlin and Vienna : Urban and Schwarzenberg. 1926. Pp. 825. Price M60 ; bound, M66.

THE third volume of this handsome encyclopædia, the previous volumes of which have already been favourably noticed in this JOURNAL, fully maintains

the high standard of its predecessors. A long article of over 150 pages, from the pen of Professor Marburg, deals with vestibular, cerebellar, and ponto-cerebellar tumours, and is particularly useful from the viewpoint of differential diagnosis. In two tables are summarised the symptoms referable respectively to the cochlear and the vestibular divisions of the eighth nerve when the site of the tumour is, in turn, in one of the following five positions, viz., ponto-cerebellar angle, cerebellum, ventricle, pons and medulla, and corpora quadrigemina. We do not remember having seen in any other textbook so useful a compendium of diagnostic data. Dr. Donk writes on the operative treatment of cerebral tumours, and Dr. Sgalitzer on the radiotherapy of ear affections, inclusive of tumours of the posterior fossa. The difficult problems concerned with dyskinesis and dystonia and their relation to disorders of the cochleo-vestibular apparatus are discussed at considerable length by Dr. E. Pollak, whose article is of much interest at the present time, even if to some extent inconclusive and open to criticism.

Among other monographs to which attention may be directed are those on miners' nystagmus, seasickness, on problems arising out of flying, on neuroses and psychoses in which cochlear and labyrinthine phenomena are prominent.

No detailed examination of this third volume is possible in the space at our disposal, but in our opinion it constitutes an admirable addition to the literature of aural neurology and in its presentment of modern systematised knowledge easily surpasses previous publications dealing with the same subjects. It is a fine product of the scientific and clinical activities of the Viennese school of medicine.

S. A. K. W.

Etudes de Clinique et Pathologie Nerveuse. By D. PAULIAN, Physician in Chief of the Neurological Department of the Central Hospital, Bucharest. First series. Bucharest : Imprimerie 'Cultura.' 1926. Pp. 153. Price not stated.

DR. PAULIAN has collected in this small volume a number of his papers dealing with clinical neurology and neuropathology, most of which have already appeared in foreign journals. Among them we may mention the more outstanding. One deals with the treatment of epidemic encephalitis, both in its acute and its chronic stages, by means of 'spinal autoserotherapy,' the patient receiving by lumbar puncture some 10 cc. of his own blood serum, withdrawn the day before and injected after being raised to a temperature of 56° C., a similar quantity of spinal fluid being removed. The results claimed by the author are such as to justify further experimentation with this method. Another paper records in detailed fashion the nervous complications of typhus fever, and is a useful contribution to a little known subject. Another is concerned with showing that the blood pressure of tabetics is lower than normal, that

it rises during attacks of lightning pains, gastric crises, etc., and that the prompt administration of adrenalin is particularly serviceable in cutting short these distressing conditions. A further interesting paper bears on the problem of the differential diagnosis between Parkinson's disease and states of Parkinsonism. According to Dr. Paulian there is a definite difference in sedimentation tests in the two conditions. He allows venous blood to run into a syringe which contains a five per cent. solution of sodium citrate, and transfers the mixture to a tube of some 4 mm. diameter, graduated with a millimetre scale, observing the sedimentation every quarter of an hour. In Parkinson's disease it is found to be more rapid and more abundant.

L'Organe sous-commissural du Cerveau chez les Mammifères. By Dr. KNUD H. KRABBE, Copenhagen. With 17 plates. Copenhagen: A. F. Host & Son. 1926. Pp. 83. Price not stated.

AT the posterior end of the third ventricle the ependymal lining on the under surface of the posterior commissure differs in form and dimensions from that covering the rest of the neighbouring ventricular surface. It is thicker and arranged in several layers, its cells are prismatic rather than cuboidal, and between it and the transverse fibres of the commissure is interposed a bed of neuroglial tissue. These differences have suggested to more than one observer the possibility that here is situated an organ, with a function, and to it the name of the 'sub-commissural organ' has been given. It is found throughout the vertebrate series and is especially prominent in certain mammals; it is recognisable in the human fœtus at an early and a late stage, but after birth it soon loses its distinctiveness and becomes altogether rudimentary. As is known, the fibre of Reissner is also found in association with the posterior commissure, to which it reaches in most mammals, but it, too, is absent in man.

Dr. Krabbe's monograph contains an account of all that is known in connection with this organ, and a record of his researches on its form in a large number of mammals, as well as in man. He also discusses possible theories of its function, concluding with great hesitation that it has conceivably a motor function relating to the circulation of the cerebrospinal fluid through the iter of Sylvius.

Methodik der Untersuchungen von Nervenelementen des makro- und makro-mikroskopischen Gebietes. By Professor Dr. W. WOROBIEW, Charkow. With 39 illustrations. Berlin: Oscar Rothacker. 1926. Pp. 130. Price RM15; bound, RM18.

FOR the anatomist and the histologist Professor Worobiew's book provides material of lively interest. By means of an ingenious technique full details of which are given, colouration and lighting being specially important, the author is able to demonstrate the structural form of nerve cells, fibres, and

ganglia in a way which, one may safely say, will be a revelation to those who are familiar only with stained microscopical sections. The author has succeeded in demonstrating the morphology of these tissues in situ, using a special binocular microscope for the purpose, so that the student can obtain a visual knowledge of nerve elements on a larger scale than is possible by mere dissection, yet without losing that sense of their continuity which ordinary microscopical methods will not allow. The beautiful illustrations provided in the book, especially, perhaps, those of nerve ganglia, seem to open a new world to the eye that has been educated only by the routine of practical anatomy and of microscopical histology. It should be said that Professor Worobiew's method is applicable to many other structures than those which concern the neurologist, as his photographs show.

The Pathology, Diagnosis and Treatment of Functional Nervous Diseases. By PAUL BOUSFIELD, M.R.C.S., L.R.C.P. London: William Heinemann. 1926. Pp. 212. Price 6s. net.

THE author bases his subject upon an energic psychological foundation and in Part I stresses the thesis that the relief of tension is a biological aim. "Pain represents the conscious manifestation of a state of tension, and pleasure the conscious manifestation of the reduction of tension." This is regarded as true both of the physical and mental worlds. Through various conscious and unconscious mental processes emotional conflicts may be engendered and result in the discharge of tension in abnormal ways, with the production of psychopathological symptoms. The various methods of psychotherapy are sketched out and their rationale discussed in Part II. Part III is devoted to the description and treatment of the common functional diseases, in which the Freudian classification and terminology are followed. Their specific pathology in the main is wisely omitted. Useful chapters dealing with the psychogenic element in disorders of menstruation and pregnancy, the sexual life, and alcoholism are included. The mental factor in asthma and mucous colitis is briefly discussed, while in conclusion the psychoanalytic treatment of so-called functional insanity is spoken of in a few pages.

Any adverse criticism can only be directed to a few points. Though hypochondria is mentioned among the actual neuroses (p. 116) it is not included in the classification or dealt with in any way. The general practioner will surely look for information on this not infrequent and ill-understood neurosis. We miss, too, any discussion on the secondary aim of a psychoneurosis, which for various reasons is of great practical import. The final chapter on functional insanity should be reconsidered, as therein are many inaccuracies and terms used loosely. Is it really true to say that "many neurotic and hysterical manifestations progress and merge into unmistakable and true dementias"? To say that dementia præcox and paranoia are the two forms of psychosis which will respond best to psychoanalysis and that many psychologists of

repute have thus cured cases, are bold statements which could hardly be scientifically verified. On investigation such cases have usually been found to have been diagnosed wrongly. One would be much more inclined to state that it is particularly in these two types of psychosis that psychoanalysis would be of little or no avail, though *paranoid* reactions which not uncommonly are met with in psychoneuroses may be cured by its means. True paranoiacs do not have hallucinations as stated. The relationship of psychoanalysis to the psychoses must be handled more tentatively, or false impressions will be made on uncritical readers be they lay or medical.

Apart from these drawbacks the book deals very well with the functional nervous disorders and is written in a clear and popular style which will make the contents all the more acceptable to a larger sphere of readers. The index shows many important omissions.

C. S. R.

Personality. By R. G. Gordon, M.D., D.Sc., M.R.C.P.Ed. London: Kegan Paul, Trench, Trubner & Co., Ltd. 1926. Pp. 802. Price 10s. 6d. To attempt within comparatively small confines a wide conspectus of the problems of human personality, physiological and pathological, or normal and abnormal, is a task in some ways as difficult as to deal with them after the manner of the encyclopædia. What strikes the reader of Dr. Gordon's attractive volume most is possibly the author's facility in reaching the heart of a problem with succinctness and clarity. There are many questions to be handled if any adequate conception of the mosaic of constituents in personality is to be gained; for example, discussion is required to determine as far as may be the view of the interrelation of mind and body best calculated, in the author's opinion, to explain personality data. This is only a preliminary problem, as it were, yet it is one of surpassing importance. In common with various writers of recent years, Dr. Gordon clearly tends to favour a hypothesis of a dynamic form of energy, obliterating to a large extent old distinctions between mind and matter, and in support of this monistic attitude he summons to his aid the principle of emergence, according to which there is no inherent inconsistency in the idea that high-grade mental processes should emerge from reflex combinations, since these combinations are more than a sum of their component units. No break of any nonplussing character can be detected in the series from the simple reflex arc, through conditioned reflexes, to those subserving consciousness and reflective thought. In the author's treatment of normal personality further consideration is given to the large groups of intrinsic and extrinsic factors moulding and modifying it, at varying levels of activity, physiological and endocrinological, and psychological. In respect of the latter, an attempt is made to evaluate the contributions of Freud, Jung, Adler, and Kempf respectively.

The second part of the book deals, on a somewhat restricted scale, with neurotic, delinquent, retarded, and dissociated personalities. If it contains no strikingly novel elements or conclusions, it is none the less readable and informative, and sound in its generalities. The main trend of the author's thought is at least as much practical as theoretical, hence the book can be recommended with confidence to the time-pressed physician, who would familiarise himself with the latest advances in our knowledge of personality problems provided they can be shown to have a bearing on his treatment of clinical material. To all such a volume of this kind should prove in an eminent degree helpful.

S. A. K. W.

Les Affections de la Colonne Vertébrale. By Professor ANDRÉ LÉRI, Paris. With a Preface by Professor PIERRE MARIE. With 115 illustrations. Paris: Masson et Cie. 1926. Pp. 526. Price 7s. 6d.

PROFESSOR LÉRI has written a particularly useful compendium of knowledge in respect of diseases of the spinal column—a subject rather apt to fall between the two stools of medicine and surgery. The book is divided into five sections, dealing respectively with congenital anomalies (of which we may specify spina bifida and its variants, sacralisation of the fifth lumbar vertebra, cervical rib, reduction in number of vertebræ); traumatic lesions; ankylosing affections; localised vertebral rheumatism; and a final chapter dealing with other conditions less definitely classifiable.

Excellent drawings (clinical, pathological and radiological) photographs, and diagrams accompany and amplify the descriptions, which are clear and accurate, if cast in a somewhat schematised mould. We do not know any volume occupying the position of this, which should fill a gap in medical literature and is sure of wide appreciation by neurologists and internists.

Les Troubles Mentaux dans les Tumeurs Cérébrales. By Dr. HENRI BARUK, Paris. With 80 illustrations. Paris: Octave Doin. 1926. Pp. 396. Price not stated.

THE question of the possible interconnection of mental symptoms with organic cerebral lesions of neoplastic origin has occupied many minds for many years, but not a few of the numerous contributions to the subject have been to a greater or less extent unsatisfactory. As a fact, the problem is beset with difficulties. In the first place, no constant relation between size of tumour and degree of mental disorder can be established, for in some cases small neoplasms are accompanied by considerable mental change, and in others large growths run their course without mental symptoms coming into the picture to any recognisable extent. Secondly, mental disorders of the most miscellaneous kind have been at one or other time described in tumour cases, involving the affective, the intellectual, or the behaviouristic side of the patient's activities, but no one can say with certitude what particular variety is to be expected

in a particular case. Thirdly, while general mental symptoms (amnesia, hebetude, irritability) have been seen as a late development in some instances, they are sometimes an early manifestation; further, they may be noted both in localisable and in unlocalisable tumours. Fourthly, the association of special mental symptom-complexes with neoplasms occupying particular cerebral sites is always subject to considerable numbers of exceptions. Once more, the actual pathogenesis of mental symptoms is shrouded in some obscurity: rise of intracranial tension, mechanical effects at a distance, local ischæmias and œdemas, and invasion of so-called associational fields, are some only of the possible factors in this connexion. Besides, we may ask whether such mental changes as arise follow solely from the presence of the tumour or take their form from the particular terrain in which it is growing; and, as if to complicate the problem more than ever, we may inquire what consitutes a mental symptom and what are to be our definitions in this respect.

All these and cognate questions are admirably handled by Dr. Henri Baruk, whose *Thèse de Paris* we have read through with much appreciation. His studies are based on some 55 cases of intracranial tumour, and he reaches his conclusions with caution and with a true scientific tendency to suspend judgment that enhances their value. We invite the neurologist and the psychopathologist to familiarise themselves with the contents of this well documented work, which also contains an excellent bibliography.

S. A. K. W.

Psychological Healing. By PIERRE JANET. Translated by E. and C. PAUL. In two volumes. London: George Allen and Unwin, Ltd. 1925. Pp. 1,265. Price £2 2s. net.

A TRANSLATION of Janet's "Les Medications Psychologiques." This work is essentially a history and criticism of the various methods of psychotherapy, and constitutes a veritable mine of information. The sections of which it is composed are of very unequal value, however, a circumstance partly ascribable to the fact that the book is largely made up of reproductions of lectures and addresses delivered at various periods, some of them being more than twenty years old.

The earlier chapters give a historical account of the development of hypnotism and suggestion from the doctrines of the magnetisers, a description of the persuasionist schools, whose tenets are termed by Janet "medical moralisation," and of the "philosophical" methods of treatment lying outside the walls of orthodox medicine, including Christian Science. In the following section treatment by "psychological economies" is considered, comprised in which are the rest and isolation methods developed from Weir Mitchell. All this is extraordinarily well done, and provides a comprehensive and fascinating account which has not been excelled.

The final chapter of the first volume deals with "Treatment by Mental Liquidation," in which Freud's theory and practice are critically reviewed. This chapter is on a very different level of achievement. The facts are incorrectly stated, the criticisms clearly prejudiced, and a failure to grasp the essentials of the Freudian position is apparent throughout.

The second volume comprises treatment by education and re-education, and other methods of a less precise kind, and concludes with a bibliography.

The translators have done their work exceedingly well, and have succeeded in preserving the charm and clarity which attaches to all Janet's writings.

Behaviorism. By JOHN B. WATSON. London: Kegan Paul, Trench, Trubner & Co. Ltd. 1925. Pp. 251.

THIS volume consists of twelve lectures written in popular style and evidently addressed to a lay audience. Those who feel that Dr. Watson has made real and permanent additions to psychological knowledge may read this work with a sense of disappointment. Too much is claimed for behaviorism: it is here represented almost as a new message to mankind, and the author appears to feel that if the behaviorist were entrusted with the education of of the young (without parental interference), the improved outlook of the coming generation would in time "make the world a fit place for human habitation." There is, of course, no reason why Dr. Watson should not visualize a millennium and work for its consummation, but it may reasonably be objected that his suggestions for the upbringing of children, though important, are not derived from behaviorism as such, but from the clinical studies of maladjusted personalities by psychopathologists such as Freud, Jung and Adler. Briefly, they are scarcely original and are such as increasingly recognised as important by parents and teachers.

These criticisms are made, not because the author's views on life in general are lacking in interest and importance, but because they may tend to deflect the attention of the reader from the objective researches on young children for which Dr. Watson is himself responsible, and which have a definite interest for the psychologist. It is hoped that his most recent investigations along these lines, an account of which is included in this volume, will be embodied in future editions of his earlier book on "Psychology from the Standpoint of the Behaviorist"—a work which the student of psychology will find more helpful as an introductory textbook than the one with which this notice is concerned.

H. D.

Hygiène Mentale. By Dr. M. POTET, Physician Specialist to the Military Hospitals, etc. Paris: E. Le Francois, 1926. Pp. 599. Price 40 francs.

THE aim of this comprehensive volume is to supply psychiatrists with an exposé of the numerous publications relating to the manifold aspects of mental hygiene. It is addressed to those commencing the study of psychiatry, medical practitioners, educationists, sociologists, lawyers and, indeed, to all those who are

interested in the evolution of the human mind. The scope of this work may be indicated by a summary of its contents. In the first part a survey is given of the development of the mental hygiene movement in various countries. The second part is devoted to an account of the various methods of psychological examination (mental tests, etc.). The principles of mental prophylaxis are outlined in the third part. In the fourth the various directions in which the principles of mental hygiene can be applied are indicated: psychic education, professional orientation and adaptation, and eugenics. The fifth part is concerned with an account of the application of the principles of mental hygiene to predisposed and abnormal individuals with special reference to the question of open clinics for early mental cases, backward and abnormal children, drug-addicts, and delinquents. A full bibliography is appended to each chapter. The book, which may be recommended to psychiatrists as a useful work of reference, includes a short introduction by Dr. Toulouse.

H. D.

An Outline of Abnormal Psychology. By JAMES W. BRIDGES. Third edition. Columbus, Ohio: R. G. Adams & Co. 1925. Pp. 236.

THIS book, the first edition of which appeared in 1919, is designed "to serve as a guide for students of abnormal psychology in the absence of a comprehensive textbook." It is essentially a classified list of abnormal phenomena, and of the chief theories which are held with regard to them. Each chapter concludes with a bibliography. As a work of reference, and as an indicator of the directions in which fuller information can be obtained concerning the various departments of psychopathology, the book should prove of considerable value.

The Clinical Examination of the Nervous System. By G. H. MONRAD-KROHN, M.D.(Oslo), M.R.C.P.(Lond.), M.R.C.S.(Eng.). Third edition. London: H. K. Lewis & Co., Ltd. 1926. Pp. 201. Price 7s. 6d.

THE third edition of Dr. Monrad-Krohn's guide to the examination of the nervous system has been slightly enlarged by the addition of accounts of the pharmacological tests of the vegetative nervous system, the pilomotor and postural reflexes, the technique of cisterna magna puncture, and the use of lipiodol injections in conjunction with *X*-ray examination. The addition of a number of original illustrations enhances the value of the book, which may be recommended to the student with the utmost confidence.

Psychotherapy. By Dr. EDWARD W. TAYLOR. Harvard Health Talks. No. 14. Cambridge, Harvard University Press. (London: Humphry Milford. Oxford University Press.) 1926. Pp. 53. Price 4s. 6d. net.

AN excellent brief resumé of the history of mental healing. Five general periods are dealt with—the stages of miracles and mysticism preceding the 18th century; the animal magnetism of Mesmer in the 18th century; the beginning

of the scientific method in hypnotism; the clinical period of Charcot and Janet; and, lastly, the analytical schools of Freud and Jung. The subject is simply but very interestingly and instructively presented. It is a pity that such a useful little work addressed to the public should be relatively expensive.

C. S. R.

Allgemeine Grundlagen der Reflexologie des Menschen. By Professor W. BECHTEREW, Leningrad. With a Preface by Professor AD. CZERNY, Berlin. With 10 illustrations and 19 plates. Third edition (German), edited by Professor MARTIN PAPPENHEIM, Vienna. Leipzig and Vienna: Franz Deuticke. 1926. Pp. 436. Price M. 26; bound, M. 29.

THE veteran Professor Bechterew gives in some four hundred pages a *revue d'ensemble* of his life-work in respect of human personality. Pointing out in some detail the shortcomings of all subjective methods of approach to personality problems, he emphasises afresh the value of objective methods, and while not here vouchsafing the reader a full description of his technique he elaborates rather the results this has furnished in all levels of neural activity, from the lowest to the highest. For him the psychical processes of the higher animals are brain-processes; all the actions of living beings can be classed as reflexes of offence and defence, consecutive to specific external stimuli and inward ruling conditions. The general thesis is maintained with an extraordinary wealth of illustration, much of which is distinctly impressive. The actual basis of Freudian hypotheses can be set out, according to the author, in terms of reflex action. Incidentally, he refuses to link his work in any way with the behaviour psychology of Watson and others, since its inception dates back to the eighties of last century, and since it is erected on wider conceptions.

Not a little of the interest of the closely reasoned argument resides in its effort to unify neurological and psychological mechanisms. So far from conceding the existence of unbridgeable gaps between the physical and the physiological, and between the latter and the psychological, Professor Bechterew holds the view that "neuropsychical energy" underlies all manifestations, physiological and psychological, and that it itself is but a special form of that physical energy which is all-pervading.

The volume will repay close perusal.

Hints to Probationer Nurses in Mental Hospitals. By RICHARD EAGER, M.D., Medical Superintendent, The Devon Mental Hospital. Second Edition. London: H. K. Lewis & Co., Ltd. 1926. Pp. 88. Price 1s. 6d. net.

THIS small volume, which we noted in its first edition, has evidently fulfilled a useful purpose. In this edition there are small and unimportant changes. Mental nurses undoubtedly should have some insight into psychology, but we

think it a pity that they should be taught that "brain is the organ of the mind"; that "mind is the process of thinking"; and that "it is the delicate structure of the brain that is deranged in patients suffering from mental disorder." These are narrow and somewhat inaccurate statements, and a poor foundation will thereon be laid for further conceptions of mental functioning. The terms "unconscious" and "subconscious" are loosely and at times wrongfully used. Much of McDougall's teaching is in evidence, but we fear that an endeavour to explain manic-depressive psychosis by reference to the instincts of self-abasement and self-assertion will not help. Many other minor criticisms might be made, but we have no wish to undervalue the worth of these pages, which should not only prove instructive to the probationer but stimulate to further learning.

C. S. R.

The Medical Insurance Agency

Members of the Medical Profession who wisely seek the aid of the Agency when taking out Insurance Contracts enjoy the following advantages:—

1. **THEY RECEIVE EXPERT AND UNBIASED ADVICE FREE OF ANY CHARGE OR OBLIGATION.**
2. **THEY HELP THEMSELVES AS THEY RECEIVE A REBATE ON THE PREMIUMS PAYABLE.**
3. **THEY INDIRECTLY HELP THE MEDICAL CHARITIES AS ALL SURPLUS FUNDS ARE DEVOTED TO THESE CAUSES.**

The Agency was founded in 1907 and has up to the end of 1925 :—

Saved to the Profession over £18,000
Contributed to the Charities ,, £12,500

Special features :

LIFE ASSURANCE in all forms.
MOTOR CAR INSURANCE. A special Policy for Medical men only.
FIRE AND HOUSEHOLD POLICIES. Comprehensive up-to-date Contracts etc., etc.

Write to the Agency on any Insurance Problem,
BRITISH MEDICAL ASSOCIATION HOUSE,
Tavistock Square, London, W.C. 1.

THE AGENCY exists to PROTECT YOUR INTERESTS and SAVE YOUR MONEY.